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THE RELATION OF DERMATOLOGY TO GENERAL MEDICINE*

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IT IS my pleasant duty to welcome you to this, the fourth meeting of our section, and I should indeed be ungrateful to its members if I did not look on this duty as an honor, and express to them my thanks and appreciation. It is also gratifying to note the presence of so large a number of men from other branches of medicine; the section is sensible and appreciative of your interest. At the time when its creation was proposed some question arose as to the advisability of adding another section to the already long list, but some of us felt that dermatology had earned the right to that recognition. We realize that your approval and aid at that time made its organization possible, and your growing attendance at its meetings has helped to confirm our belief that it would serve a useful purpose.

Dermatology recognizes its relation to all the other branches of medicine, and there are sound and valid reasons why this should be so. Anatomically, the skin and its appendages are of the same general structure, are controlled by the same nervous system, are nourished by the same blood supply, and have the same susceptibility to the effect of toxins and infections as do all the other organs and tissues of the body. Physiologically, the skin is not only the heat regulator and physical protector of the body, but is also the principal site of the production of its protective antibodies. Also, its function as one of the great secreting, excreting and absorptive organs brings it into reciprocal relation with all the others.

Thus, as one of the essential organs of the body, the skin not only is subject to its own special diseases, but also exhibits sympathetic manifestations in one form or another of disease in other organs, as well as shares to some extent in the well-being or ill-being of the body as a whole. Observation indicates that the relation of dermatology to general medicine is somewhat closer than is its relation to the other specialties.

It may help toward a better understanding of this relation if I review some of the steps by which American dermatology, during its comparatively short life as a specialty, has won popular approval by its practical achievements, and professional respect by its contributions to the common fund of medical knowledge. It began its separate existence with the inheritance of a group of diseases believed to be limited to the skin, which was considered to be a separate organ. They were named and classified according to their visible appearance and gross anatomic forms, with little or no knowledge of their connections or relations. This highly artificial conception, with its distorted viewpoint and meaningless nomenclature, could serve only as a handicap to scientific progress. This attitude, and the prejudice which then existed against specialism in general, offered a stern challenge, which the sponsors of dermatology as a separate specialty accepted and successfully met, building it into a specialty which could no longer be ignored. From time to time such important advances as the determination of the changes in the structural elements of the skin accompanying known morphologic changes, the effects of bacterial invasion, the relation of external contacts to altered skin conditions, and so on opened new avenues of approach, enlarged our understanding of disease processes, widened our etiologic horizon, and formed a background for the revolutionary discoveries of the past quarter century, especially in the fields of biochemistry and biophysics, which by focusing our

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attention on cell reactions under the influence of hostile contacts have given us a wholly new conception of the meaning of disease.

If dermatology has benefited as a result of this broader outlook, so too has general medicine. If medicine has aided dermatology by providing an enlarged etiologic background, dermatology has reciprocated by furnishing medicine with new and valuable methods for the detection of internal disorders. The skin has been rightly spoken of as a mirror or reflector of changing body conditions, and medicine has profited by the utilization of this function in its various phases. It is true that a large proportion of dermatoses are of local origin and limitation, and except for such influences as variations of resistance and susceptibility, have little or no proved relation to internal medicine. On the other hand, it is no less well known that in many others such a relation can be definitely shown to exist. Those of the latter group, with their many and devious ramifications, may link the skin with disease of any other organ of the body and invade every field of medicine.

These ailments may be subdivided into two minor groups. The first of these includes such diseases as tuberculosis, cancer, syphilis and leukemia, in which the skin manifestations are caused by the same etiologic factors, show the same histological pictures as the associated internal trouble, and may be considered as a part of the same general process. In the second, the symptomatic group, the cutaneous picture is not a counterpart of the parent trouble, which may be either organic or functional. It is more apt to be an atypical or distorted reflection which challenges further investigation, and its relation can be recognized and correctly interpreted only as we have learned to read the signs. These skin changes may be among the first evidences of visceral disease or dysfunction, and their recognition may give the physician his first warning of some hitherto unsuspected and perhaps serious internal trouble. To the patient, a rash means that something is wrong, and he is likely to seek early relief for it, while paying little attention to the equally important but to him less alarming malaise or vague internal discomfort. Whether in a situation like this he first visits a dermatologist or a general physician, an early consultation often proves to be useful and constructive, in that it may help to solve what is frequently a puzzling problem, and afford him an opportunity to receive treatment at a time when it may be most effective. In this important field of co-operative investigation, dermatology has contributed a generous share, as it has in the study of the effects of emotional and neurogenic factors, not only on the skin itself but also through sympathetic action on other organs. In order to be able to recognize these relations, it is necessary that the dermatologist retain at least the essentials of his early medical knowledge. In fact, it is almost axiomatic that the more one knows of general medicine, the better dermatologist he proves to be.

Another example of how the skin serves as a reflector of changed internal conditions is the well-known fact that variations of resistance and susceptibility of the body as a whole, or of any of its organs, frequently cause corresponding changes in cutaneous reactivity. This fact, coupled with the skin's ready visibility, makes it the ideal immunological testing ground of the body. One needs only to recall the many skin tests now in common usage to appreciate the value of this important skin function to general medicine. And the end is not yet, for there can be little doubt of the future discovery of tests which will be equally helpful in the detection of other diseases and abnormal conditions. This vital branch of medical research originated in the field of dermatology, and its development was carried on there, only to be adopted later, and further elaborated in some instances, by other branches of medicine. An illustration of this is our present knowledge of the condition known as allergy, which began with a study of its cutaneous reactions.

The ready accessibility of the skin, and the ease with which bacteriological and histological examinations could be made there, led to its early selection as the site where pathologic changes and the effects of bacterial invasion could be studied to the greatest advantage. It is there, too, that inoculations, cultures, biopsies, staining methods and many other diagnostic refinements can best be utilized. As has so often been said, the skin is the only organ in the body where the processes of inflammation, infection, malignancy, atrophy and so on can be studied in detail under natural conditions. Because of this, it is probable that clinical observation and pathological differentiation are more complete and elaborate in the case of the skin than in that of any other organ. Also, because it may afford the first actual evidence of beginning or early active stages of disease, as in cancer and many infections, the skin offers an ideal opportunity not only for early diagnosis, but also for studying the evolution of disease processes as applicable to other organs. This is still another way in which the study of the skin has done much to enlarge our understanding of diseases and their relations, a service which is sure to render continued aid in the solution of medical problems, and one which offers to dermatology what is perhaps its most interesting outlook.

Again, it is well known that our knowledge of irritations as the one proved cause of cancer was gained by observing its effect on the skin.

A not unimportant contribution of dermatology to general medicine is its assistance in the diagnosis of local skin diseases, which so often present confusing and rapidly changing pictures. The number of primary lesions are limited, but their variations are many and perplexing. Their repetition, and the multiplicity of designs and color schemes created by their combinations, are what make the diagnosis of skin diseases so interesting and difficult, and mark it as the peculiar forte of dermatology; familiarity with its vagaries and seeming contradictions requires long and painstaking study, for which the general practitioner can seldom spare the time. He is often inclined to smile at our hair-splitting and complicated methods, but when necessity calls for their unaided use they seem less amusing. Differential diagnosis is just as essential in dermatoses as it is in diseases of other organs, and neglect or inability to make it is too often the undoing of one's therapeutic efforts. It is only when one knows what the trouble is that he can visualize its lesions, and select the medication best suited to combat them. We have all seen such purely local conditions as scabies or pityriasis rosea being treated for suspected digestive or other internal troubles. And even ill-chosen local applications may by irritation increase discomfort, prolong duration, with resulting economic loss, and make later diagnosis difficult. Nowhere in the entire field of medicine can routine treatment be relied on to cure undiagnosed disease, yet dermatology has always received generous assistance of this type from many sources.

In the field of therapeutics dermatology has been no laggard. It has discovered, tested and approved many new and valuable agents now in common use, which have largely replaced the older empiric ones; these include the arsenicals, bismuth, Enesol, the colloidal preparations and many others. External applications have also been greatly improved. The present widespread medical use of radium, x-ray, ultraviolet light and many other physical aids had its inception in the knowledge acquired by dermatologists through employing them on the skin, where their possibilities were demonstrated and a trail was blazed for their application in other fields.

Our present knowledge of syphilis and its treatment has developed in the field of dermatology, and some of us still feel that the dermatosyphilologist is still the expert best qualified to differentiate it from the other cutaneous and membranous manifestations, any one of which it may simulate or complicate. The same tendency to simulation is seen in the later involvement of other organs by syphilis. Indeed there are few diseases which it may not ape, nor any field of medicine which it may not invade, at some time during its long and devious course, especially if the patient marries and has children. One cannot escape the conviction that, except in the case of certain individuals who accept treatment only under supervision, the present tendency to systematize treatment of such cases is less conducive to the patient's welfare than was the earlier individualized approach, with its bond of personal interest, which did so much to relieve the patient's perplexity, regulate his routine of life and preserve his self-respect.

It is no boastful prompting which leads us to declare that dermatology as a specialty needs no defense, because it has justified the faith of its founders, and has reached a stage of efficiency which compares favorably with that of other specialties; that it has demonstrated its usefulness to both the public and the medical profession, and that any interference with its continued existence and growth would be detrimental to the whole field of medicine. In the roster of its membership may be found the names of men who occupied or still occupy prominent places in the forefront of medical progress, who have found in it a field worthy of their earnest efforts, and who have given freely of their time and strength to its advancement. And I know of no greater proof of its continued interest and vitality than the way in which the younger dermatologists are striving to maintain its high standards and traditions.

With this background of practical achievement, one cannot regard with indifference the tendency on the part of some medical schools to curtail the amount of separate instruction in dermatology and syphilology, on the ground that students should devote time to the study of the basic medical sciences, leaving the specialized part of their training to optional postgraduate work. This may have its theoretical advantages, but as we all know, most graduates go into general medicine, and only a small proportion feel that they can spare the time and expense necessary for much postgraduate work beyond their internship. Many of them locate in smaller communities and work under conditions which oblige them to assume the care of skin diseases. For this reason alone, — and there are others, — it would seem highly advisable that undergraduates should continue to receive adequate instruction in this field, at least to the extent that they shall acquire a working knowledge of the commoner dermatoses. For the

same reasons we cannot look with favor on the conviction expressed in some quarters that neither the standard of medicine nor the public welfare would be adversely affected if dermatology ceased to exist as a specialty and again became a part of general medicine.

I have tried to call to your attention a few of the many ways in which the study of the skin and its functions has been helpful to general medicine and to the other specialties; I have attempted to show that it has won for itself a definite place in the field of medicine, which should be maintained, and that recognition of the problems incident to these relations and co-operation in their study will continue to promote progress, not only in our own field but in all medicine. As Pusey* has recently said, "Dermatology has a field. It has its borderlines, which are not at all points distinct and may merge into other fields. . . . But its great area is well defined, and we possess it not by right of possession but by right of discovery and useful occupation."

I realize that I have said nothing that is not common knowledge, and that has not been said better before. My only defense is that it is fitting that we pause for a moment from time to time, and recall to our minds some of the simple and vital steps by which dermatology has reached its present status; these steps have been too dearly won to be lightly cast aside, and forgotten in the daily routine of our busy lives.

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*Pusey, W. A.: The field of dermatology. Arch. Dermat. & Syph. 33:987-993, 1936.

LUPUS ERYTHEMATOSUS DISCOIDES*

Its Present Status With Regard To Etiology and Treatment

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THE increasing incidence of lupus erythematosus attested by recent reports, together with a voluminous literature, much of which is confusing, justifies an attempt to arrive at some definite conclusion as to the present status of the etiology and treatment of the disease.

Lupus erythematosus was first described by Biett in 1828 under the name of erythema centrifuge; in 1845 Hebra discussed it under the heading of seborrhea congestiva; and in 1851 Cazenave gave it its present name. All three writers referred to the form now designated as discoid or chronic. In 1872 Kaposi described the type now designated as acute or subacute disseminate. Since then much has been written concerning the various types and their relation to each other, as well as the etiology and treatment.

It is generally accepted that there are four main clinical types of lupus erythematosus: the chronic discoid or fixed, the chronic disseminate, the subacute disseminate and the acute disseminate. A failure in the early reports to indicate clearly the type under discussion has caused considerable confusion; this criticism can also be made of some of the more recent reports. The present paper deals exclusively with the discoid or fixed type. This form occurs chiefly on the face (where it characteristically attacks the bridge of the nose and the flush areas of the cheeks, the so-called butterfly outline), ears, mucous membranes and scalp. The early lesion is an erythematous, slightly elevated macule covered by adherent grayish-brown scales; it gradually increases to form the discoid plaque, the center of which is depressed and lighter in color. In the absence of scales there are seen patulous follicular orifices, which contain dry, horny epithelial plugs. Involution and cure may occur without trace or scar, either spontaneously or with therapy. More commonly, however, scarring takes place. These scars are superficial, flat, smooth, pliable, slightly depressed and white; their surface is sometimes stippled, and it may even be telangiectatic. On the scalp the scars are more sclerotic than elsewhere and produce alopecia. The course of the disease is chronic, afebrile and benign, in contrast with the disseminate forms, which are acute in onset, and are associated with fever and a multitude of pathologic findings, and which are only too frequently fatal.

The findings and opinions hereafter set forth are based on the study and follow-up of 122 consecutive and unselected cases of lupus erythematosus discoides treated at the Massachusetts General Hospital between March, 1930, and December, 1937, together with an estimate of the literature and a comparison with other results. Twenty-six more cases were seen, but are not included because of inadequate follow-up.

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DISTRIBUTION OF CASES

The series consisted of 67 females and 55 males. This greater incidence in females is true of all reports, the ratio in many being much greater than ours. All the patients were white. Lupus erythematosus is not common in the Negro, and Cummer[1] was able to gather only 30 such cases from the literature.

The age incidence, depicted in Table 1, was about the same for both sexes, except that more females were found in the second decade. It is noteworthy that there was no patient under ten years of age. This was also the experience of Davidson and Birt.[2] Cummer[3] collected from the literature 176 cases in which the patients were under sixteen; none of them were under ten and the greater number were of the disseminate type. Denzer and Blumenthal[4] reported a case of the disseminate type in a girl of seven. Wright[5] reported a case of the discoid type in a girl of five.

Table 1. *Age at Onset of Disease.*

AGE	MALES	FEMALES	TOTALS
0-9	...	...	...
10-19	3	12	15
20-29	16	20	36
30-39	17	13	30
40-49	11	15	26
50-59	6	4	10
60-69	1	3	4
70-79	1	0	1
80	...	...	...
Grand Totals	55	67	122

Recent advances in spectroscopic studies and the demonstration of various metals in the role of possible etiologic agents[6] of dermatological diseases suggested an analysis of the occupations of the group, shown in Table 2. From it may be drawn the conclusion that, except as may be correlated to other findings, occupation is of little significance.

Table 2. *Occupations.*

Actor	1	Nurse	1
Bookkeeper	2	Plumber	1
Boxmaker	1	Railroad worker	1
Carpenter	3	Restaurant	1
Chauffeur	4	Rubber worker	1
Clerk	4	Sailor	1
Electrician	1	Salesman	2
Farmer	1	Shoe worker	4
Fireman	1	Student	5
Garage worker	1	Surveyor	1
Housewife	38	Telephone operator	1
Jeweler	1	Toolmaker	1
Laborer	4	Watchman	1
Machinist	4	Weaver	1
Molder	1	Unemployed	11

The duration of the disease at the time of the first visit to the clinic varied from two weeks to thirty years. There were 46 cases of less than one year's duration, 15 of one year, 18 of two years, 22 of three to five years and 21 of more than five years (Table 3). As later shown in Table 14, the duration at the first visit was an important factor in the probability and duration of cure. Finally, that this report represents a true cross section of the white race is evidenced by an analysis of the different national groups. (Table 4.)

Table 3. *Duration of Disease at First Visit.*

DURATION	NO. OF CASES	DURATION	NO. OF CASES
2-3 wk.	2	4 yr.	4
1 mo.	3	5 yr.	11
2 mo.	7	6 yr.	2
3 mo.	7	7 yr.	7
4 mo.	5	8 yr.	2
5 mo.	3	10 yr.	2
6 mo.	11	12 yr.	1
7 mo.	4	15 yr.	1
8 mo.	1	18 yr.	1
9 mo.	1	19 yr.	1
10 mo.	2	20 yr.	1
1 yr.	15	22 yr.	1
2 yr.	18	24 yr	1
3 yr.	7	30 yr.	1
		Total	122

Table 4. *National Groups.*

FOREIGN-BORN (37 CASES)			
Ireland	7	Sweden	2
Italy	7	Nova Scotia	2
Poland	3	Scotland	2
Russia (Jewish)	3	Belgium	1
Greece	3	China (American)	1
Canada (French)	2	Newfoundland	1
Finland	2	England	1
NATIVE (85 CASES)			
Massachusetts:		Maine:	
American	26	American	2
Irish	24	Swedish	1
Italian	12	Scotch	1
Scotch	2		— 4
French	2	Connecticut:	
Jewish	2	American	1
Portuguese	1	Swedish	1
	— 69		— 2
New Hampshire:		Rhode Island:	
French	2	American	2
American	1	Irish	1
Irish	1		— 3
	— 4	Pennsylvania:	
New York:		American	2
French	1		— 2
	— 1		

ETIOLOGY

The agents variously designated as the causative factor in the production of lupus erythematosus may be listed as follows: tuberculosis, foci of infection, toxemia, external physical agents (trauma, sunlight, ultraviolet rays, x-rays), internal physical agents (porphyrin, lead), allergy and nonspecific tissue response.

Tuberculosis

From the time of Cazenave,[7] who regarded lupus erythematosus as a variant of lupus vulgaris, up to the present a considerable amount has been written to prove or disprove tuberculosis as an etiologic factor. The early writers such as

Riehl regarded the disease as a true tuberculosis. Later, with the development of the theory of a toxin due to the tubercle bacillus, the French school, led by Hallopeau, Besnier and others, suggested a tubercle toxin as the etiologic agent.

An analysis of the earlier literature supports the thesis that much of the evidence for a tuberculous background is purely presumptive. Weiss and Singer[8] found tuberculosis mentioned in only 64 out of 225 cases of lupus erythematosus collected from the English literature. Keil[9] culled from the reported cases 125 cases with postmortem examination. Only 20 per cent showed active or possibly active tuberculosis; moreover, many of the cases were of the disseminate type. It may even be said that in most cases the association is casual; considering the ubiquity of tuberculosis and the fact that few people escape infection prior to adult life, it is only natural to expect to find evidence of tuberculous infection at postmortem examination of people dying from other and unrelated causes. Lastly, as indicated by MacLeod,[10] a number of the early reported cases cited as indicating the association of the two diseases are based on errors in diagnosis, due chiefly to confusing the disease with lupus erythematoides (Leloir), a true tuberculosis cutis clinically resembling lupus erythematosus and seen where tuberculosis is common.

On the other hand, Cannon and Ornstein[11] inoculated guinea pigs with biopsy material from 5 cases of lupus erythematosus and produced both macroscopic and microscopic lesions of tuberculosis, pointing to the tubercle bacillus as an etiologic agent. The association with tuberculosis is not easily downed, many writers assuming the attitude that "one must not easily put tuberculosis aside." As late as 1935 Krenn[12] stated, "The percentage of positive cultural findings is much higher in lupus erythematosus than in any other form of cutaneous tuberculosis."

In this series the patients were questioned as to tuberculosis contact, family history and signs and symptoms pointing to possible infection earlier in life. They were examined for physical signs, and where possible, x-rays of the chest were obtained. Except in a few cases tuberculin tests were not done. Tuberculosis in some form was encountered in 16 cases (Table 5). There were 4 cases with a history of contact with tuberculosis and negative x-rays, also 4 cases with a negative history but with roentgenological findings consistent with tuberculosis. It is notable that 50 per cent were cured* with treatment. There were 4 cases associated with tuberculous adenitis and negative x-ray findings; all were cured with treatment — curiously enough, 3 with bismuth and 1 with gold. Only 2 cases were associated with active pulmonary tuberculosis.

Table 5. *Cases with a Clinical Finding of Tuberculosis.*

CASE NO.	SEX	HISTORY	X-RAY OF CHEST	TYPE OF TREATMENT	RESULT OF TREATMENT
10	F.	Husband in sanatorium with active pulmonary tuberculosis.	Negative	Gold	Failure
57	F.	Sister died of tuberculosis. Patient was exposed.	Fibrosis of right lower lung; no active tuberculosis.	Gold	Cure
113	F.	Grandmother died of tuberculosis.	Negative	—	Insufficient
121	F.	Sister in sanatorium with active pulmonary tuberculosis.	Negative	Gold	Cure
22	F.	Negative.	Healed tuberculosis ?	Bismuth	Cure
36	M.	Negative.	Pleurisy, left base, and old hilum glands	Gold	Cure
108	F.	Negative.	Old hilum glands	Gold	Marked improvement
4	F.	Negative.	Healed tuberculosis; no activity.	Gold	Failure
25	F.	Tuberculous adenitis 11 years previously.	Negative	Bismuth	Cure
69	M.	Tuberculous adenitis 20 years previously.	Old lung abscess	Bismuth	Cure
65	M.	Tuberculous adenitis 20 years previously.	Negative	Bismuth	Cure
101	F.	Developed a tuberculous node while under treatment for lupus erythematosus. Excised.	Negative	Gold	Cure
8	M.	Tuberculous peritonitis 10 years previously. Subsequently died of this cause.	Negative		Failure
35	F.	Healed tuberculous keratitis. Positive tuberculin test.	Negative	Gold	Cure
44	M.	Developed incipient tuberculosis while under therapy for lupus erythematosus.	Original x-ray negative	Gold	Moderate improvement
76	F.	Active pulmonary tuberculosis. Onset 4 years previously. Sanatorium case.	Active tuberculosis	—	Insufficient

These cases represent 13 per cent of the cases; if however, the contacts and the x-ray cases are subtracted, this percentage is reduced by about half. An analysis of the tabulation confirms the impression that the association of tuberculosis with lupus erythematosus is casual and of no direct etiologic import.

Foci of Infection

The significance of focal sepsis in the etiologic mechanism of lupus erythematosus was first emphasized by Barber and other English writers,[13]

*By "cure" is meant disappearance of the disease without recurrence up to the time of the present summation of cases.

who isolated strains of streptococci from intestines, teeth and tonsils, and correlated their eradication with improvement in the skin condition. In this country Hartzell[14] called attention to this phase of therapy. Since then, a long and formidable list of possible foci of infection has been assembled, and it is now a generally accepted routine to search for and eliminate them. Although most of the work has been directed against the streptococcus,[15] other organisms have also been implicated. Hopkins and Burky[16] reported a more intense reaction to staphylococcus in patients with lupus erythematosus as a group than in any other dermatosis.

Table 6. *Teeth as Foci of Infection.*

	NO. OF CASES
Possible focus	41
X-ray	33
Clinical	8
X-ray negative	35
Clinically negative	21
Absent	17
Not studied (controls)	8
Total	122

If we accept Billings's[17] definition of focal infection as "a dissemination of bacteria and toxic products from the focus of infection," it is but natural to expect also the formulation of a long list of possible foci such as that offered by Ayres and Anderson.[18] However, in a congested outpatient department and in a world in which one can add finance as a focus of irritation, if not of infection, an extensive and elaborate search is not always feasible. In the present series a complete physical examination, as well as a rigorous search for foci, always preceded treatment, but save in cases of obvious trouble elsewhere the writer must plead guilty to the criticism of not going beyond the tonsillar pillars in the active removal of foci.

Table 7. *Results of Correction or Extraction of Teeth as Sole Foci of Infection.*

	NO. OF CASES
Without additional treatment:	
Cure	1
Failure	4
With additional treatment:	
Cure	9
Marked improvement	6
Moderate improvement	2
Failure	6
Total	28

Teeth. Every case was examined in the Dental Clinic and treated as recommended. There were 41 cases in which the teeth were found to be a possible focus (Table 6), and in 28 of these they were the only obvious demonstrable focus. The results of removal with and without additional treatment are shown in Table 7. It is to be mentioned that 6 of the cases classed in Table 7 as moderately improved or failures had insufficient treatment. Three cases had recurrences traced to new abscesses; of these, 1 had one, 1 two and 1 three recurrences. There were also 3 cases in which the teeth were found to be an obvious focus and removal was advised but refused. Two of them failed to improve without therapy; they were then treated with gold sodium thiosulfate and showed marked improvement.

Table 8. *Tonsils as Foci of Infection.*

	NO. OF CASES
Possible focus	30
Surgically absent	22
Clinically negative	62
Not studied (controls)	8
Total	122

Tonsils. Every case was examined in the Nose and Throat Clinic for possible foci. The statistical findings are shown in Tables 8 and 9. The effect of removal is shown in Table 10.

Table 9. *Sinuses as Foci of Infection (X-ray Examination).*

	NO. OF CASES
Negative	31
Cloudy or thickened	8
Pansinusitis	1
Cyst of floor of antrum	3
Epithelioma	1
Not studied (controls)	8
X-ray not indicated	70
Total	122

It seems therefore on purely statistical evidence that, where the teeth or tonsils alone were involved, removal of teeth as foci was of much greater importance than that of tonsils. However, the number of failures listed in Table 7 suggests the possibility of incomplete eradication of the possible foci. This is substantiated by an analysis of 13 cases in which both the teeth and tonsils were implicated (Table 11).

Table 10. *Results of Removal or Non-Removal of Tonsils as Sole Foci of Infection.*

Tonsillectomy done		9
With additional treatment:		
Cure	3	
Marked improvement	2	
Failure	2	
Without additional treatment:		
Failure	2	
Tonsillectomy not done		8
With other treatment:		
Cure	5	
Marked improvement	2	
Failure	0	
Without other treatment:		
Failure	1	
Total		17

In these cases there were no failures when both foci were eradicated, whereas when the teeth alone were removed failure resulted in 3 of 4 cases. Eight cases were used as controls; that is, they were deliberately not checked for possible foci. Four of these cases were cured with other treatment; 2 improved markedly and 2 resulted in failure.

Table 11. *Results of Treatment in Cases in Which Both Teeth and Tonsils Were Foci of Infection.*

Teeth only removed		4
With additional treatment:		
Cure	1	
Failure	3	
Teeth and tonsils removed		3
With additional treatment:		
Cure	1	
Marked improvement	1	
Moderate improvement	1	
Neither removed		6
With other treatment:		
Cure	2	
Improvement	1	
Failure	1	
Without other treatment:		
Failure	2	
Total		13

Trauma

Trauma as a factor in the etiologic mechanism of lupus erythematosus has been often mentioned in the literature, chiefly by Smith,[19] Cummer and Stein. In our series there are 7 cases which developed directly on the site of injury to the skin. Two cases were preceded by a razor cut, 3 by frostbite, 1 by an oil burn, and in 1 the lupus developed in a tattoo mark. Five of these cases responded well to treatment and 2 were failures.

Sunlight

The harmful influence of sunlight upon erythematous lupus was noted by the earliest observers. With[20] in 1921 reported the first instance of this disease as the result of artificial ultraviolet light, and Civatte[21] in 1926 called attention to the effect of light rays on toxic substances contained in the skin as a possible causative factor. Since then the relation between sunlight and lupus erythematosus has been the subject-matter of many excellent articles.[22]

The actual mechanism of the influence of sunlight on this disease has not been established. The rarity of the condition in Negroes has already been mentioned. It is probably cutaneous pigment which protects the Negro against the erythema caused by the sun. Pardo-Castello[23] found erythematous lupus no more frequent in Cuba than in the United States. Hasselmann[24] saw only 5 cases in six years' experience in the tropics (the Philippine Islands). Ludy and Corson[6] found hematoporphyrin in the urine in 10 cases of lupus erythematosus discoides out of 27 examined. They mention, however, that Goeckerman and Tulipan did not find it in their cases. Brunsting and Nomland[25] found porphyrinuria in a chronic case of long duration. It has been pointed out, however, that this may well have been an end result rather than a causative agent. Recently Turner and Obermayer[26] expressed the belief that sensitization to porphyrin plays little or no part in the production of lesions of hydroa aestivale. They suggest differentiating hypersensitivity of the skin and altered reaction of the skin. Ludy and Corson also performed spectroscopic studies on biopsy material from cases of lupus erythematosus, and found lead present frequently enough to suggest it as a contributory cause of the disease. They believe that the presence of lead in these cases was analogous to the presence of a porphyrin and produced the same result.

The existence of a sensitizing substance in the skin has led to attempts to offset the effects of sunlight by the use of various substances, notably quinine. Davidson and Birt[2] used quinine both locally and internally as a desensitizing agent, on the theory that the drug might in some way change the reaction of the skin to the ultraviolet band in sunlight.

Some time ago (1920) Stokes[27] suggested that cutaneous sensitization from a pyogenic focus might serve as a secondary etiologic factor in the production of lupus erythematosus with tuberculosis as a primary cause, or possibly the reverse. In this way he regarded the condition as a toxic or allergic manifestation of tuberculous and streptococcal infection. More recently he[28] emphasized the predisposing effect of light on the susceptibility to pyogenic infection and the reverse effect.

In the present series there were only 19 in which sunlight played an important role. Ten cases had a positive history of onset shortly after sunburn; 4 cases, not originally associated with exposure to light, were aggravated by exposure to the sun. Five of the 10 cases originally said to have followed sunburn had recurrences following re-exposure to sunlight. Seven of the 10 had no demonstrable foci; and with treatment 4 were cured, 2 were markedly improved, and 1 was a failure. There were 5 cases in which patients received ultraviolet therapy before coming to the clinic and had been made worse by it.

THERAPY

Prior to the introduction of aurotherapy there was no one specific method of attack in the treatment of lupus erythematosus. In view of the present concept of the disease, the older methods

of local destruction must be confined to cases having but one or two lesions of long duration and without tendency to spread, or to employment as an aid in refractory cases.

Gold

When European investigators directed their attention to the study of tuberculosis through autherapy, it followed that dermatologists began to employ gold compounds in the treatment of lupus erythematosus because of its supposed tuberculous background. In America this form of therapy was popularized by Schamberg and Wright,[29] who used gold sodium thiosulfate and Throne et al.[30] who used Triphal. The history of gold therapy and discussions of its physiological action, chemistry, toxicity, reactions, contraindications and the comparative values of the various gold compounds, have been amply described in the literature[31] and do not call for repetition here. It may be stated, however, that autherapy represents a nonspecific form of medication, and that in general the reactions to and the distribution of gold salts on injection resemble very closely those of other heavy metals.

In the series here presented gold was used exclusively in the form of gold sodium thiosulfate. Sixty-eight patients were treated, and received 1546 injections, or a total of 78.3 gm. The injections were given intravenously at weekly intervals. The initial dose was 25 mg.; in some cases this was reduced to 10 mg. The average weekly dose was 50 mg. It was found that in some of the seemingly refractory cases an increase of the dose to 75 mg. increased the therapeutic response; however, in the truly obstinate cases increase of dosage to 100 or 200 mg. made little difference in the response to therapy. The largest single dose administered was 200 mg.

Improvement and cure was not in any way proportional to the total number of injections. Many of the cures came after a few injections, whereas others required many. One case was resistant to one hundred injections for a total of 11.84 gm.; he also failed to respond to other methods of treatment. Lashinsky[32] suggested that treatment be stopped if no improvement has taken place after twenty injections. The present group does not warrant such a conclusion. The type and degree of involvement of lupus erythematosus, varying as they do, force the conclusion that the number of injections must be determined on the basis of the individual case. It may well be that many of the reported failures with gold have been due to premature cessation of therapy.

The untoward reactions to gold therapy in this series were as follows: deaths, 0; a tendency to become disseminate, 6; joint pains, 2; jaundice, 2; albuminuria, 2; dermatitis and pruritus, 5. Such reactions have been well studied and reported in detail by Driver and Weller[33] and by Throne, Kingsbury and Myers.[34]

An attempt to study the blood picture was made in 25 cases. The red cells changed very little during treatment; in 3 cases the count fell below the original level; in 5 cases it increased; in the other cases it remained on the same approximate level. The initial leukocyte count in most of the cases tended toward the lower limits of normal. This finding is in agreement with the statistics of Weiss and his associates.[35] However, except in those cases which tended to disseminate, the leukocyte count during therapy followed no definite pattern and varied to such a degree that no definite conclusion could be drawn. The differential count in general showed a relative increase in the lymphocytes as therapy proceeded, but too few counts were made to allow a true evaluation. No determinations of the rate of red-cell sedimentation were done.[36]

Therapeutic results were obtained as follows: 31 cases (46 per cent) were cured; 11 markedly improved; 6 moderately improved; 20 (29 per cent) were failures. These figures compare well with the statistics reported by others. Towle[37] in 1931 found 319 American cases of lupus erythematosus with 51 per cent cures and 7 per cent failures, and 101 foreign cases with 45 per cent cures and 6 per cent failures. Wright[5] in 1936 reported 76 cases with 34 per cent cures and 12 per cent failures. It may well be concluded that the greater percentage of failures in Wright's series and in that here presented are due rather to more exacting criteria for cure and a longer period of observation for possible recurrence.

Recurrences took place in 14 (21 per cent) of the cases, with multiple recurrences in 5. Wright reported 13 (17 per cent) relapses in his 76 cases. Callaway and Stokes[38] in an excellent analysis of the reasons for relapse report 22 recurrences (74 per cent) in 31 cases.

Bismuth

The use of bismuth in the treatment of lupus erythematosus originated in France. Lortat-Jacob and Legrain[39] mentioned it in connection with several cases of the disease in association with syphilis (1927). Two years later Nicolas, Lacassagne and Rousset[40] employed bismuth in cases not associated with syphilis and reported good results. In England, MacKenna[41] advocated bismuth therapy both locally and by injection. It was his belief that the improvement was due to the ability of bismuth to prevent the tissues from

reacting normally to the toxins or bacteria which caused the disease. In 1934 Smith[42] reported 12 cases treated with bismuth salicylate, resulting in 33 per cent cures, as compared with 42 per cent cures in 12 cases treated with Sanocrysin. He advocated bismuth as the metal of choice because "it is less toxic; it does not aggravate an existing tuberculosis as gold may do; there is less chance of focal reaction; it is simple and cheap to administrate." In this country the use of bismuth has been reported by Madden, Abramowitz, Wise and Sulzberger, and Diasio.[43]

Forty-three of our cases were treated with bismuth for a total of 1155 injections, given intramuscularly at weekly intervals. The preparation first used was iodobismuthate of quinine, but it was found that bismuth salicylate in oil seemed to be giving better results, and it was therefore used in the entire series so as to permit uniform observation. With bismuth, as with gold, it was found that the number of injections required to effect an improvement or cure varied widely with each case. It may be stated, however, that where bismuth proved efficacious it could be so in as few injections as was gold. There were 6 cases of unfavorable reaction to bismuth therapy: bismuth line, 3; albuminuria, 2; and rash, 1.

The therapeutic results were as follows: cured, 14 cases (32 per cent); markedly improved, 11; moderately improved, 2; failures, 16 (37 per cent). Recurrences took place in 5 cases (12 per cent). Except for the statistics quoted from Smith no reported comparative figures are available.

Calcium

The beneficial effect of calcium observed in several cases of the acute disseminate type led to its use in a group of the cases comprising this report. Such treatment of lupus erythematosus has not, so far as could be ascertained, been previously reported. Downing and Blumenfeld[44] and Lain[45] do not mention it in their reports on calcium therapy. Schwartz and Levin[46] examined the calcium content of the blood in 4 cases of lupus erythematosus and found it to be normal in all.

Twenty-seven of our cases were treated with calcium in the form of calcium levulinate. The injections were given intravenously once or twice a week, using 10 cc. of a 10 per cent solution. Except for a temporary heat wave, and in some cases a slight dizziness lasting but a few minutes, there were no true untoward reactions. The therapeutic results were as follows: cured 5 (18 per cent); markedly improved 6; moderately improved 2; failure 14 (52 per cent). There were 3 recurrences (11 per cent).

COMPARISON OF THERAPEUTIC VALUES

An analysis of Table 12 indicates that from a comparative viewpoint bismuth is as valuable as gold in the treatment of lupus erythematosus, and that calcium is far inferior to both. This is much more clearly demonstrated by a group of 36 cases in which all three were used, an opportunity thus being given to compare them in any one case (Table 13). The fact, however, that the three agents were all able to effect cures emphasizes a point previously made that their action is not of a specific nature. Better to test this point a group of cases has been recently started on therapy with colloidal manganese, and the results thus far seem to hold promise for the future although not enough time has elapsed to allow evaluation or comparison.

Table 12. *Comparative Therapeutic Results of Gold, Bismuth and Calcium.*

No. of Cases	Substance	Cured		Markedly Improved	Moderately Improved	Failure		Recurrence		Insufficient Treatment
		No.	%	No.	No.	No.	%	No.	%	No.
68	Gold sodium thiosulfate	31	45.6	11	6	20	29.4	14	21.0	7
43	Bismuth salicylate	14	32.5	11	2	16	37.2	5	11.6	4
27	Calcium levulinate	5	18.5	6	2	14	51.8	3	11.0	4
15	No treatment	1		0	0	14		0		0

It has already been demonstrated that the dosage and number of injections required to effect improvement or cure was not a constant, but varied greatly in the individual cases. The duration of the disease at the time treatment was instituted did influence the probability and duration of cure (Table 14). There were 50 cases in which a cure was effected; of these, 38 were of less than three years' duration at the time of the first visit and 16 of one year's duration or less. There were 12 cases with a five-year cure, all of them of two years' duration or less, and 8 of them had had the disease less than one year.

The therapeutic value of calcium, bismuth and gold must lie in their ability to act as alterative agents acting upon the skin directly or upon one of the factors involved in the etiologic complex of lupus erythematosus. That they do not influence the entire complex is evidenced by the occurrence of recrudescences during therapy and by recurrences traced to such definite factors as

abscesses of teeth, intercurrent infection and exposure to sunlight.

CONCEPT OF LUPUS ERYTHEMATOSUS

The essential morphologic and clinical features of lupus erythematosus discoides are well known and generally agreed upon. The etiology of the disease is, however, still much disputed and not fully understood. It may be said with safety that it is certainly not of tuberculous origin. The findings in the series presented with regard to focal sepsis, while definitely indicating its importance, leave much to be desired. Sunlight is certainly an important factor, as are other agents. Must we agree with Sulzberger[47] that lupus erythematosus is not an etiologic but rather a morphologic diagnosis, or may we hold that it is an example of the ability of tissue to respond uniformly to various pathogenic agents?[48]

There is one very important phase of lupus erythematosus that must not be lost sight of in any commentary on the disease; that is the fact that under certain circumstances it may become disseminate and acute. What then is the relation of the different types to each other? There is a tendency for internists[49] to regard the acute and subacute disseminate types as independent diseases. The multiplicity and variety of clinical manifestations,[50] together with the severity, poor

Table 13. *Comparison of Results from Gold, Bismuth and Calcium Therapy.*

	NO. OF CASES
Gold and bismuth	18
Gold better than bismuth	7
Bismuth better than gold	4
Bismuth equal to gold	5
Both failures	2
Gold and calcium	8
Gold better than calcium	5
Calcium better than gold	1
Both failures	2
Bismuth and calcium	5
Bismuth better than calcium	3
Calcium better than bismuth	2
Gold, bismuth and calcium	5
Failures	5
Total	36

prognosis and lack of scarring in the morphologic picture, are interpreted as lending support to this thesis. If, however, it is borne in mind that alleged exciting agents such as sunlight, trauma, tuberculosis and foci of infection are found in the case histories of the acute as well as the chronic forms, and that under certain circumstances these same agents may cause the chronic form to become acute and disseminate, one may well dispute the theory of two distinct diseases.

Wise[51] has expressed the opinion that the different forms of lupus erythematosus are in reality but phases of the same condition, an opinion vigorously defended and advocated by Keil.[52] On the basis of considerable pathological study of both types, Goeckerman and Montgomery[53] conclude that although it is often possible to distinguish between the two types, a sharp line of demarcation cannot be drawn because the same changes are present in both, varying only in degree.

The therapeutic results obtained in the present report allow the conclusion that gold therapy

Table 14. *Relation of Duration of Disease at Time of First Visit to Cure.*

DURATION AT FIRST VISIT	LENGTH OF CURE 1 YR.	2 YR.	3 YR.	5 YR.
1 mo.				1
2 mo.		1		
3 mo.		1	3	2
4 mo.				1
5 mo.				1
6 mo.	1	1	1	2
8 mo.				1
1 yr.	2	1	5	2
2 yr.	2	3	5	2
3 yr.	2	1		
4 yr.		3		
5 yr.	1		1	
6 yr.				
7 yr.		1		
8 yr.	1	1		
12 yr.		1		
Totals	9	14	15	12

is not specific or indispensable and that bismuth is as valuable as gold. Moreover, the observation that other therapeutic agents such as calcium and manganese can cause cures, although to a lesser degree, points to the conclusion that they act as alteratives, an action limited and influenced by other factors, as is evidenced by the number of failures and a marked tendency of the disease to recur.

It is therefore deemed justifiable to offer the hypothesis of an etiologic complex, with the constitutional background of the patient the keyboard upon which the exciting agent may strike and call forth a responsive note in the guise of lupus erythematosus. This note may then be dampered by factors as yet unexplained and result in the chronic discoid form of the disease, or it may become one of a series of overtones and produce the clinical picture of acute disseminate lupus erythematosus.

SUMMARY AND CONCLUSIONS

A series of 122 consecutive and unselected cases of lupus erythematosus is presented, and analyzed from etiologic and therapeutic standpoints. The results are compared with others reported in the literature.

Tuberculosis is not a direct causative agent; in most instances the evidence is presumptive and the relation casual.

Foci of infection should be searched for and

eliminated. Focal infection is, however, but a part of a larger etiologic complex.

The influence of sunlight on erythematous lupus is definitely harmful, although its mode of action has yet to be explained.

Gold therapy is not specific or indispensable.

Bismuth is an excellent therapeutic agent, as valuable as gold and less dangerous.

Both gold and bismuth act as alteratives.

The best results are obtained when treatment is started early and is kept up even in the face of recurrence.

The various clinical types are but phases of the same disease.

The hypothesis is offered that lupus erythematosus is the product of an etiologic complex acting on the constitutional background of the individual patient.

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REFERENCES

1. Cummer, C. L.: Etiology of lupus erythematosus; occurrence in the negro. Arch. Dermat. & Syph. **33**:434-445, 1936.
2. Davidson, A. M., and Birt, A. R.: Quinine bisulfate as a desensitizing agent in the treatment of lupus erythematosus. Arch. Dermat. & Syph. **37**:247-253, 1938.
3. Cummer, C. L.: Lupus erythematosus in infancy and in childhood. Arch. Dermat. & Syph. **24**:999-1032, 1931.
4. Denzer, B. S., and Blumenthal, S.: Acute lupus erythematosus disseminatus. Am. J. Dis. Child. **53**:525-540, 1937.
5. Wright, C. S.: Ten years' experience in the treatment of lupus erythematosus with gold compounds. Arch. Dermat. & Syph. **33**:413-433, 1936.
6. Ludy, J. B., and Corson, E. F.: Lupus erythematosus; increased incidence, hematoporphyrinuria and spectroscopic findings. Arch. Dermat. & Syph. **37**:403-416, 1938.
   Gaul, L. E., and Staud, A. H.: Clinical spectroscopy; study of biopsy material taken from patients receiving gold sodium thiosulphate. Arch. Dermat. & Syph. **28**:790-794, 1933.
7. Cazenave, M.: Lupus érythémateux. Ann. d. mal. de la peau **3**:297-300, 1851.
8. Weiss, R. S., and Singer, J. J.: The relation of lupus erythematosus discoides to tuberculous infection. Am. J. M. Sc. **155**:528-540, 1918.
   Goeckerman, H. W.: Is lupus erythematosus discoides chronicus due to tuberculosis? Arch. Dermat. & Syph. **3**:788-801, 1921.
9. Keil, H.: Relationship between lupus erythematosus and tuberculosis; critical review based on observations at necropsy. Arch. Dermat. & Syph. **28**:765-779, 1933.
10. MacLeod, J. M. H.: Lupus erythematosus: some observations on its etiology. Arch. Dermat. & Syph. **9**:1-12, 1924.
11. Cannon, A. B., and Ornstein, G. G.: The tubercle bacillus as an etiologic factor in lupus erythematosus. Arch. Dermat. & Syph. **12**:691-699, 1925.
12. Krenn, O.: Lupus erythematosus and tuberculosis. *The 1937 Year Book of Dermatology and Syphilology*. 736 pp. Chicago: The Year Book Publishers, 1937. P. 224.
13. Barber, H. W.: A case of acute lupus erythematosus. Brit. J. Dermat. **27**:365-371, 1915. A case of lupus erythematosus associated with streptococcal infection of the tonsils. *Ibid.* **31**:186-193, 1919. Protein sensitization and focal sepsis in the etiology of certain skin affections. Guy's Hosp. Rep. **71**:385-414, 1921.
   Low, R. C.; Logan, W. R., and Rutherford, A.: A fatal case of lupus erythematosus; with autopsy. Brit. J. Dermat. **32**:253-262, 1920.
14. Hartzell, M. B.: Lupus erythematosus and focal infection. Arch. Dermat. & Syph. **2**:441-446, 1920.
15. Shaffer, L. W.: Role of the streptococcus in etiology of pemphigus, lupus erythematosus and erythema group of hematogenous dermatoses. J. Michigan M. Soc. **36**:292-297, 1937.
16. Hopkins, H. H., and Burky, E. L.: Increased reactivity of the skin to staphylococcus toxin in patients with lupus erythematosus. Arch. Dermat. & Syph. **33**:1060, 1936.
17. Billings: quoted by Ayres and Anderson.[18]
18. Ayres, S., Jr., and Anderson, N. P.: Focal infection in dermatology. Arch. Dermat. & Syph. **34**:421-431, 1936.
19. Smith, D. K.: External irritation as a factor in the causation of lupus erythematosus discoides. Arch. Dermat. & Syph. **14**:547-549, 1926.
   Stein, A. A.: Lupus erythematosus on sites of operative trauma. Urol. & Cutan. Rev. **40**:654-656, 1936.
20. With, C.: An eruption of lupus erythematodes on the breast and upper arm, caused by carbon light-baths in patient with lupus erythematodes on the face. Förhandl. vid Nord. dermat. förening. **4**:130-133, 1919. Abstr. in Zentralbl. f. Haut. u. Geschlechtskr. **2**:87, 1921.
21. Civatte, A.: Research on etiology of erythematous lupus during 20 years. Ann. de dermat. et syph. **7**:465-485, 1926.
22. Bechet, P. E.: Excessive solar and phototherapeutic irradiation, causative factor in certain diseases of the skin. Arch. Dermat. & Syph. **29**:221-227, 1934. Lupus erythematosus disseminatus, with extension of the disease after exposure to actinic light. *Ibid.* **13**:701, 1926. Acute disseminate lupus erythematosus following sunburn. *Ibid.* **26**:538, 1932.
   MacKee, G. M.: Ultraviolet therapy in dermatology. J. A. M. A. **98**:1553-1561, 1932.
   Feit, H.: Light as an exciting agent in lupus erythematosus and other dermatoses. J. M. Soc. New Jersey **24**:226-228, 1927.
23. Pardo-Castello, V.: discussion of Cummer, C. L.[1]
24. Hasselmann, C. M.: Lupus erythematosus (discoideus) in the tropics; first report of cases from Philippine Islands and investigations on occurrence of Langhans' giant cells. Arch. Dermat. & Syph. **29**:585-596, 1934.
25. Brunsting, L. A., and Nomland, R.: Lupus erythematosus (telangiectatic type); epithelioma (basal cell); hematoporphyrinuria. Arch. Dermat. & Syph. **27**:1035, 1933.
26. Turner, W. J., and Obermayer, M. E.: Studies on porphyria; case of porphyria accompanied with epidermolysis bullosa, hypertrichosis and melanosis. Arch. Dermat. & Syph. **37**:549-572, 1938.
27. Stokes, J. H.: discussion of Hartzell, M. B.: Lupus erythematosus and focal infection. Arch. Dermat. & Syph. **2**:441-446, 1920.
28. Stokes, J. H., and Callaway, J. L.: Pyogenic relapse and sensitiveness to light in certain dermatoses; influence of factor of intercurrent infection. Arch. Dermat. & Syph. **36**:976-986, 1937.
   Stokes, J. H.: discussion of Engman.[29]
29. Schamberg, J. F., and Wright, C. S.: The use of gold and sodium thiosulphate in the treatment of lupus erythematosus. Arch. Dermat. & Syph. **15**:119-137, 1927.
30. Throne, B.; Clark, A. S., and Van Dyck, S.: Treatment of lupus erythematosus with gold compounds (sodium salt of aurothiobenzimidazol carboxylic acid). New York State J. Med. **27**:1064-1075, 1927.
31. Throne, B.: Lupus erythematosus: a clinical study. Arch. Dermat. & Syph. **12**:33-40, 1925.
   Monash, S., and Traub, E. F.: Modification of therapy with gold compounds in lupus erythematosus: preliminary communication. Arch. Dermat. & Syph. **24**:110, 1931.
   Jones, J. W., and Alden, H. S.: Modification of therapy with gold compounds in lupus erythematosus; preliminary communication. Arch. Dermat. & Syph. **28**:544, 1933.
   Wigley, J. E. M.: Some observations on the deposition of gold in the skin. Brit. J. Dermat. **44**:69-76, 1932.
   Pillsbury, D. M., and Kulchar, G. V.: Gold dermatitis limited to depigmented skin, with theoretical consideration of its etiology. Arch. Dermat. & Syph. **27**:36-48, 1933.
32. Lashinsky, I.: discussion of Greenhouse, C., and Spinner, G. F.: Lupus erythematosus. Arch. Dermat. & Syph. **25**:1174-1176, 1932.
33. Driver, J. R., and Weller, J. N.: Untoward results from the use of gold compounds: report of a fatal case. Arch. Dermat. & Syph. **23**:87-109, 1931.
34. Throne, B.; Kingsbury, J., and Myers, C. N.: Unusual clinical manifestations following intravenous administration of gold compounds. Arch. Dermat. & Syph. **25**:494-507, 1932.
35. Weiss, R. S.; Lane, C. W., and Bagby, J. W.: Effect on the leukocytes of therapy with gold preparation. Arch. Dermat. & Syph. **35**:1074-1086, 1937.
36. Tulipan, L., and Director, W.: Red cell sedimentation in dermatology: preliminary report. Arch. Dermat. & Syph. **27**:759-765, 1933.
37. Towle, H. P.: Present status of gold therapy. New Eng. J. Med. **204**:487-489, 1931.
38. Callaway, J. L., and Stokes, J. H.: Relapse in lupus erythematosus after treatment with sodium gold thiosulfate. Arch. Dermat. & Syph. **37**:627-630, 1938.
39. Lortat-Jacob, L., and Legrain: Lupus érythémateux du visage chez une hérédosyphilitique; guérison par le bismuth. Bull. Soc. franç. de dermat. et syph. **34**:19, 1927.
40. Nicolas, J.; Lacassagne and Rousset, J.: Le traitement du lupus érythémateux par les injections de sels de bismuth. Paris. med. **71**:62-66, 1929.
41. MacKenna, R. M. B.: Treatment of lupus erythematosus with bismuth. Lancet **1**:178-180, 1930.
42. Smith, A. G.: Gold and bismuth in the treatment of lupus erythematosus. Brit. J. Dermat. **46**:399-410, 1934.
43. Madden, J. F.: Chronic discoid lupus erythematosus: results of treatment with bismuth. Arch. Dermat. & Syph. **27**:359, 1933.
   Abramowitz, E. W.: Generalized erythroderma from bismuth in a patient with a fixed type of lupus erythematosus of the face. Arch. Dermat. & Syph. **25**:1115-1118, 1932. Discussion of Wright.[5]
   Wise, F., and Sulzberger, M. B.: Gold and bismuth in the treatment of lupus erythematosus. *The 1935 Year Book of Dermatology and Syphilology*. 736 pp. Chicago: The Year Book Publishers, 1935. P. 451.
   Diasio, F. A.: The treatment of lupus erythematosus with special reference to value of gold and bismuth compounds. M. J. & Rec. **137**:375-378, 1933.
44. Downing, J. G., and Blumenfeld, A.: Effect of calcium in pruritic skin affections; with especial reference to calcium gluconate. New Eng. J. Med. **204**:250-253, 1931.
45. Lain, E. S.: Calcium metabolism and therapy in dermatology. South. M. J. **29**:626-630, 1936.
46. Schwartz, H. J., and Levin, O. L.: The calcium content of the blood in various diseases of the skin: based on an analysis of over three hundred cases. Arch. Dermat. & Syph. **10**:544-550, 1924.
47. Sulzberger, M. B.: A case for diagnosis (lupus erythematosus). Arch. Dermat. & Syph. **37**:138-140, 1938.
48. Highman: discussion of Hartzell, M. B.: Lupus erythematosus and focal infection. Arch. Dermat. & Syph. **2**:441-446, 1920.
49. Baehr, G.; Klemperer, P., and Schifrin, A.: Diffuse disease of peripheral circulation usually associated with lupus erythematosus and endocarditis. Tr. A. Am. Physicians **50**:139-155, 1935.
50. Belote, G. H., and Ratner, H. S.: The so-called Libman-Sacks syndrome: its relation to dermatology. Arch. Dermat. & Syph. **33**:642-664, 1936.

Engman, M. F., Jr.: Early acute lupus erythematosus. Arch. Dermat. & Syph. 35:685-697, 1937.
Jarcho, S.: Lupus erythematosus associated with visceral vascular lesions; a series of autopsied cases. Bull. Johns Hopkins Hosp. 59:262-272, 1936.
Keil, H.: Relation between "systemic" lupus erythematosus and a peculiar form of thrombocytopenic purpura. Brit. J. Dermat. 49:221-237, 1937.
Mook, W. H.; Weiss, R. S., and Bromberg, L. K.: Lupus erythematosus disseminatus. Arch. Dermat. & Syph. 24:786-829, 1931.
Scholtz, M.: Lupus erythematosus acutus disseminatus hemorrhagicus. Arch. Dermat. & Syph. 6:466-475, 1922.
51. Wise, F.: Lupus erythematosus bullosus or pemphigoides; report of two cases with discussion of "Senear-Usher syndrome." M. J. & Rec. 134:227-231, 1931.
52. Keil, H.: Conception of lupus erythematosus and its morphologic variants, with particular reference to "systemic" lupus erythematosus. Arch. Dermat. & Syph. 36:729-757, 1937.
53. Goeckerman, W. H., and Montgomery, H.: Lupus erythematosus; an evaluation of histopathological examinations. Arch. Dermat. & Syph. 25:304-316, 1932.

Discussion

Dr. E. Lawrence Oliver, Boston: Dr. Tolman has presented a very instructive paper. It was my privilege to see and follow many of the patients included in this report while I was on duty at the Massachusetts General Hospital, hence the paper is of special interest to me.

The teeth and tonsils are always possible foci of infection which may and probably do play a part in the etiology of some cases of lupus erythematosus. Of course, foci of infection may exist in many other parts of the body, such as the appendix, the gall bladder, the prostate, the fallopian tubes, the sinuses, the lymph nodes and possible hidden abscesses anywhere in the body. As Dr. Tolman has stated, unless there was obvious trouble elsewhere it was necessary in this study to limit the search for foci to the teeth, tonsils and sinuses. The figures given indicate that the chances of cure are greater after abscessed teeth have been removed. It is significant that the figures indicate that infected teeth are of much greater importance than infected tonsils, although the number of cases is too small to draw any definite conclusions from them.

In regard to the etiology of lupus erythematosus, the trend, in this country at least, is away from tuberculosis as a prime factor and toward the theory that toxins of other bacteria from focal infections are more influential. This theory is very likely the correct one, but before we discard the idea that tuberculosis or its toxins may be closely associated as an etiologic factor in this disease, we should be sure that the focal infections in teeth, tonsils or elsewhere are not of tuberculous origin. Have we examined sufficiently the tonsillar tissue removed in such cases, so as to be certain that no tuberculosis is present? Have we inoculated guinea pigs with this material to make sure that some of these tonsils are not tuberculous? Is it beyond the bounds of possibility that some of the apical abscesses in teeth are of tuberculous origin? Some of these abscesses, I believe, occur in the sac at the root of a tooth. This sac has a serous lining somewhat analogous to the lining of the pleural and peritoneal cavities, which are subject to infection with the tubercle bacillus. Would it not perhaps be worth while to inoculate guinea pigs with the pus from root abscesses in order to rule out the possibility of tuberculosis? The fact that other bacteria are found in these abscesses does not necessarily eliminate the possibility of a tuberculous infection. Until all these tests are made we should not be too sure that tuberculosis is a negligible factor in the etiology. I am not championing the cause of tuberculosis as the important etiologic factor in this disease, but I feel that we must have more proof against this theory before we can be justified in discarding it entirely.

That exposure to sunlight and lupus erythematosus are in some way related, in certain cases at least, has been noted by various observers during the last century. In view of this it seems strange that the incidence of the disease is not greater in the tropics. Perhaps the explanation of this lies in the fact that residents of the tropics usually take precautions against overexposure to sunlight.

Dr. Tolman is to be congratulated on the high percentage of cures in his series of cases. There is no doubt that the introduction of gold therapy in lupus erythematosus aroused great interest all over the world. The results in many cases have justified its wide use. My impression has been that if a patient did not improve after twenty treatments with gold salts it was not worth while to continue them, but this report does not bear out this theory, so it would seem that we are justified in giving longer courses of treatment. That gold treatment is not always innocuous must always be remembered. For this reason it is good to hear that a high percentage of cures was obtained with intramuscular injections of bismuth salicylate. The use of injections of calcium levulinate is interesting, but the results indicate that it will prove much less valuable than treatment with gold or bismuth salts. The group of cases now under treatment with colloidal manganese is of considerable interest.

In regard to the etiology of lupus erythematosus, I feel that we are still very much in the dark.

A Physician: Has a trial of x-ray therapy been made in the treatment of lupus erythematosus.

Dr. Tolman (closing): X-rays should seemingly be of value; but for reasons as yet not accounted for, discoid phases of the disease may suddenly become disseminated, and any such treatment must be used with caution. Furthermore, the actinic effect of x-rays is not too far removed from that of sunlight, and can produce the harmful effects on the skin that sunlight does. Moreover, cases have been reported in which following x-ray exposure the lesions became disseminated and appeared in places removed from the areas to which the x-ray was applied.

ARE PATCH TESTS OF REAL VALUE IN DERMATOLOGY?*

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THE past fifty years have produced notable advances in medicine. Dermatologists have not lagged in contributing to them, especially as regards the fields of allergy, mycology and industrial medicine. The most important issue today is not a general agreement that eczema is of endogenous or exogenous origin, or both, or a recognized classification of the cutaneous manifestations of allergy, but a continuance of the investigative work which is simplifying the maze of descriptive morphology in which dermatologists have been wandering.

Contact tests and patch tests, also known as eczema or epidermic tests, have played no small part in this work by revealing causative factors of contact dermatitis, that is, dermatitis venenata. This condition, as defined by James C. White[1] in his classic monograph, *Dermatitis Venenata,* includes those inflammations of the skin which are produced by the direct action of irritating agencies, externally applied: "The nature of these agents is various, and their number is very large. They belong to the vegetable, animal and mineral worlds, and to other classes of matter not so easily defined." This paper will attempt to evaluate patch tests in the light of their history, their various applications and their interpretation.

HISTORY

Contact tests have probably been used for centuries. Boys have tested themselves boastingly or their playmates mischievously with irritating plants. In 1904 Nestlar[2] left the juice of the primula on his skin for three days, and also attached part of the plant to the skin with sticking plaster, letting it remain there for several hours at a time; after fourteen days he suffered a severe reaction, sensitization taking place through the unbroken skin. In 1921, Low[3] performed a similar experiment on himself. At that time he believed it possible to produce an immunity by this method, and applied small doses of *Primula obconica* to his brother's scarified arm for nine days. Three weeks later he applied the plant to the latter's intact skin, and four days after that a severe eruption appeared, which lasted for four weeks. Repeated testing showed that he had produced a generalized and probably lifelong sensitivity in both himself and his brother.

Low investigated the cutaneous sensitiveness of workers with morphine and its compounds, strychnine and other alkaloids. An opium worker had a dermatitis resulting from his occupation which cleared up in eight weeks, at which time Low applied a paste of crude opium to the patient's forearm. Six hours later the man had an extensive dermatitis, which kept him in a hospital for four weeks. Low also tested other workers with various compounds, and suggested that such tests might enable an employer to tell what substances his employes could handle with safety. At a later date, Bloch,[4] by applying an extract from the primrose to the unbroken skin, sensitized all of a selected group of normal, previously non-sensitive persons, and showed that the reaction was caused by a simple organic, nitrogen-free product.

The patch test was introduced by Jadassohn[5] in 1894, was developed by Bloch,[4] and was employed in animal experimentation in America by Markley[6] in 1920.

METHODS OF APPLICATION

The patch test is a simple procedure. The suspected irritant, covered with a piece of linen and a still larger section of cellophane, is fastened to the skin with strips of adhesive plaster; or a piece of linen soaked in a solution of the irritant is substituted for it and similarly applied. The cellophane keeps in the moisture, and being larger than the linen helps to differentiate the central reaction of the test substance and that which may arise from the plaster. If the patient is highly sensitive to adhesive, or if a solid object such as a hairpin is to be tested, the test object or substance should be covered with a moisture-proof material and held to the skin by cotton bandages. The substance should be left on the skin for twenty-four hours, unless there are subjective symptoms.

A method for multiple tests on the forearm suggested by Biederman[7] consists in applying small squares of blotting paper impregnated with the test solution; these are separated by narrow strips of adhesive and covered with waxed paper, the

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plaster being so arranged that as many as twenty tests can be made at once on each forearm.

A simple and effective patch test which Reynolds[8] states has proved adequate for insecticides, bakelite powder, soda solutions and oil involves the use of a cotton pad, held in place by collodion, under which is a piece of sterile cotton which holds the suspected substance against the skin.

Volatile substances may be tested simply by exposing the skin to the vapor, as Biederman[9] demonstrated by having a patient hold his forearm over the open mouth of a bottle of gasoline.

The drop method is recommended by Wedroff.[10] The test chemical, in varying concentrations, is dissolved in 90 per cent alcohol and dropped on the skin. The alcohol evaporates and the residue remains. One drop of pure spirits is used as a control. In testing aqueous solutions, it may be well to clean the skin with ether. Lacquers and paints should be applied directly with a brush. Powders should be applied in dry and moistened states. Appropriate solvents, such as weak acids or alkalies, should be employed; those soluble in fat may be tested in oils or vaseline. Greases and ointments may be applied directly to the skin. Schwartz[11] has suggested the use of a stock fluid approximating perspiration in composition, with which he moistens the skin or the patch-testing material. By adding a weak acid or alkali he has obtained positive reactions where negatives had resulted from ordinary tests.

Shelmire and Black[12] have extracted forty plant oils by the simple means of soaking the plants in ether for twenty-four hours, and then pouring the ethereal extraction into an open vessel and allowing it to evaporate. In applying these oils to the skin no covering is placed on the sites, which are numbered with silver nitrate. Controls with the various stock solutions or solvents must always be used. If other persons are employed as controls, they should have normal skins, and the test should be made on a skin area corresponding to that of the patient.

Urbach[13] recommends that negative tests be repeated on healed areas. Concentration below the stimulus necessary to produce reactions in normal individuals must be utilized.

Engelhardt and Mahler[14] were able to show that patients with an elevated temperature and increased sweating were especially sensitive to external irritants applied in patch tests. When there was increased sweating without heightened temperature, the effect was not noticeable.

In general, careful history-taking is essential in the use of the patch test; in industrial investigations, two separate histories should be taken, with an interval between them to enable the patient to recall additional details. In this way, an etiologic factor may be disclosed which requires no further corroboration, and the field of investigation may be narrowed.

As noted above, the tested substance should be left on the skin for twenty-four hours in the absence of subjective symptoms. At the end of that time the reaction is noted and graded according to Bloch's[15] classification, which is as follows:

1. Redness
2. Redness, swelling and papules
3. Intense redness, swelling, papules and vesicles
4. Large, confluent bullae
5. Desquamation, oozing and even necrosis

Besides these reactions there may be mild or severe flare-ups of the active or healed dermatitis, with a rapid extension of the pre-existing eruption.

RESULTS AND INTERPRETATIONS

The interpretation of the results of patch testing requires experience, knowledge and judgment. Sulzberger[16] has said that for the patch test to be useful it must be properly applied and properly read; the reaction must be correctly evaluated; the test must be employed with full knowledge of its limitations; and it must be utilized only when and where indicated by the given case. He and Wise[17] in 1931 limited the value of the tests to cases of dermatitis venenata and drug eruptions of the eczematous type, such as those from mercury or arsphenamine; they held that their usefulness had not been proved in chronic lichenified eczema, neurodermatitis, seborrheal dermatitis, urticarial eruptions, angioneurotic edema and non-eczematous drug eruptions. They warned against the use of primary irritants, and asserted that the elimination of the substance or substances giving positive tests, or desensitization with them, would cure the condition.

Peck and Salomon[18] believe that patch testing is more important for children with eczema than is scratch testing. This statement applies with equal force to those with neurodermatitis.[19] On the other hand, Obermayer and his associates,[20] employing the same technic as did Peck and Salomon, found that the patch test was of no utility in neurodermatoses, but was the method of choice for determining the cause of eczema — dermatitis venenata.

Elimination of the irritant is successful, but desensitization has so far proved of little or no value except in the use of plant oils; and even with these, despite enthusiastic reports, the results have been disappointing to many dermatologists; furthermore, attempts at desensitization have occasionally caused serious reactions such as der-

matitis exfoliativa. Brunsting and Anderson[21] were not enthusiastic over their results in ragweed desensitization. Urbach states that in fixed drug eruptions a positive test may be obtained if it is carried out within the area of erythema. Other authors have been unable to corroborate this.

Diphenyl compounds, tars, greases and oils may give an acneform reaction to epidermic tests, especially in persons suffering from acne vulgaris. A 1:30 dilution of trichophytin, old undiluted tuberculin and 25 per cent potassium iodide or potassium bromide, in hydrous wool fat, all give similar reactions.[22]

Ingram[23] discovered by making patch tests that 4 per cent of normal individuals showed an idiosyncrasy to phenylenediamine, reacting on the first contact. He was impressed with the large proportion of cases in which reactions were delayed as long as twenty-four days. He wondered whether such cases were indicative of a mild degree of sensitization or were the result of the development of sensitization in the epidermis after application of the chemical. Tested subjects, he believed, must be kept under observation for at least a month in order to secure full and accurate results. Such late reactions are difficult to explain, but reactions after four days are more apt to be sensitizations produced by the test substances. Biederman[9] demonstrated by continued testing of a person sensitized to gasoline that he could produce a generalized eruption, although only the forearms were exposed. This patient was sensitized to three types of gasoline.

In the cases mentioned above one wonders whether there was not a so-called unknown or eczematous tendency, and whether, if the patient had been tested with a series of similar possible irritants, there would not have been a series of positive reactions. False positive reactions may be due to the employment of substances which injure normal skins; for example, in testing with waxes and resins of unknown composition a normal person should be used as a control. The use of routine tests may show a positive reaction to substances not contacted; frequently a worker thinks that chrome is present in his work when in fact he is exposed to no such chemical. If the skin is tested too near the affected area, false positives also result. The same is true if the affected area is tested before complete recovery—a procedure which has been known to cause false negatives in severe dermatoses such as occur after exposure to butesin picrate.[24]

False negatives may also come from testing with too weak a concentration. The area tested may not have been properly selected; more negative reactions result from patch tests applied to the back than from those applied to the anterior or inner aspect of the arm, or to the chest in the axillary line. The patient may be in a state of hyposensitivity at the time of testing. Physical agents and their influences cannot be reproduced, nor can trauma or the action of vital agencies.

Considerable experimental work has been performed with plants by means of patch testing. Spain and Cooke[25] found that 65 per cent of patients over eight years of age were sensitive to the active principles of poison ivy. Straus[26] found that a poison ivy paste applied to 41 adults produced a positive reaction in 76 per cent. He patch-tested 119 newborn infants with a similar paste with completely negative results; some of the infants in addition to the skin application were fed a solution of poison ivy for prophylaxis. After a suitable interval, 48 of the patients were retested by the patch method and a positive reaction was obtained in 35 cases, or 73 per cent. The skin was generally sensitized. Straus concluded that the insensibility to poison ivy heretofore observed in children under three was due not to a lack of mechanism but to a lack of previous contact or of sufficient contact. If the sensitizing contact is adequate, infants will react with a positive response to patch tests in approximately the percentage of adults.

Field and Sulzberger[27] found that a patient who had been sensitized to poison ivy experimentally reacted when patch tested in from twenty-four to seventy-two hours, depending upon the concentration of the extract applied. The sensitization reached such a degree that this individual, who had previously failed to react to concentrations as high as 1:100, later reacted strongly to a 1:1,000,000 dilution of the extract. There were also variations dependent on the time of testing and the section of skin tested. As a result, these authorities warn against drawing definite conclusions from one or a few patch tests simultaneously applied; they believe that their experiment explains certain discrepancies and variations of reactions hitherto unaccounted for, as well as false negatives.

Brown, Milford and Coca[28] tested with plant oil 40 persons subject to ragweed hay fever, 40 subject to other atopic hypersensitiveness, and 40 normal persons without individual or family atopic history. Six persons in each group reacted, showing that ragweed dermatitis is not due to the atopic excitant but to the oil itself, and that it is therefore a contact dermatitis.

Contact tests may be of value in the outlining of therapy, especially where mercury is indicated, for if a small amount is applied to the skin in an unaffected area this will reveal the presence of a

sensitivity. Schoch[29] concluded from the results of patch tests used in 8 cases of arsphenamine dermatitis that the test with neoarsphenamine may be relied on to permit, if negative, or to interdict, if positive, further arsphenamine therapy in patients who have recovered from this form of dermatitis. Cormia[30] found the patch test of no service in determining cutaneous hypersensitivity to neoarsphenamine in guinea pigs. Chargin, Sulzberger and Crowley[31] patch-tested 503 patients — 210 with and 293 without syphilis — with a 10 per cent solution of neutralized arsphenamine; 21 per cent of the latter and 17 per cent of the former gave positive reactions. Two of those with syphilis who had negative reactions later developed an arsphenamine dermatitis. None of those with positive reactions did so. These investigators concluded that the contact test did not indicate whether an arsphenamine dermatitis was or was not likely to develop.

NOTES CONCERNING TREATMENT

Before drawing conclusions from these varying interpretations of the patch test, it will be well to point out a few principles of treatment in addition to those already mentioned. It cannot be overemphasized that the problems presented to the dermatologist in modern industry are among the most intriguing in this branch of science. With the history of a specific contact, such as that with poison ivy, and an eruption occurring within forty-eight hours, dermatitis venenata presents no difficulties. But with the rapid changes in our mode of life, the variegated contacts to which we are exposed in our homes through importing both plants and animals from their natural habitats, the so-called civilized manner of clothing and adornment, and the rapidly increasing and ever-changing hazards in occupation have resulted in rebellious outbursts on the skins of numerous individuals. Contact tests frequently furnish the clue to the problems which these eruptions present.

In the first place, it is often impossible to know the chemistry of the solutions to be used in testing, and when such substances must be applied it is well to keep the patient at the office as long as possible. Volatile solvents and essential oils should be dropped on the skin; an occlusive dressing may cause a severe reaction, for many of these substances act like primary irritants upon hypersensitive individuals. Reactions with bullous formation have been seen to result in a few minutes.

Patch tests should be applied and removed by the physician. In compensation and medicolegal cases he cannot testify to the results of contact tests unless he has administered them. A reaction in order to be considered positive must have caused lesions similar to those of the eruption in question.

Not only the separate chemicals but their many combinations must be tested; that is, the patch test should be carried out in such a manner as to imitate as closely as possible the original contact. In testing cosmetics, the powder, cream and rouge should be tested separately and also in the combinations used by the patient. False negatives result from the impossibility of reproducing the friction of application. Baer[32] showed by patch testing that methyl heptine carbonate was the cause of lipstick dermatitis.

Too many closely allied chemicals should not be applied at one time, for if the patient has been sensitized to one of them, they all may cause reactions, with severe systemic complications. This is especially true of dyes; for example, a fur salesman, after repeated attacks of dermatitis and disability, was tested (when his eruption was almost relieved) by an intern with fairly large pieces of ten different types of dyed fur. In about eight hours he had an almost generalized eruption, accompanied by an uncontrollable itch which disabled him for three months. The interesting feature of this case was that on removal of the patch tests the sites were negative, although four areas later showed reactions.

Negative reactions at patch test sites, with marked surrounding reaction, should always be viewed with suspicion and not ascribed solely to the adhesive plaster; for as surgeons have known for years, reactions do occur outside of occlusive dressings that have enclosed iodine, chlorinated soda or mercury preparations. The control test should always be placed well away from the test materials, and held on by a separate strip of adhesive for the purpose of comparison.

VALUATION OF PATCH TESTING

It is evident from the investigations described in this paper that there is pronounced disagreement as to the efficacy of patch tests as applied to various cutaneous diseases. It is, however, generally agreed that this kind of test is the best medium for determining the causative factor in cases of epidermal sensitization, namely dermatitis venenata. On the other hand, when we review the sensitizations produced by experimental patch testing of human beings, and the small amount of valuable information thus derived, we wonder whether we are justified in producing groups of sensitized individuals; for, as Bloch[33] remarked after his brilliant work with primin, the human subject sensitized to primrose is for obvious reasons not very suitable for the more

exact study of a series of questions in the domain of eczema pathogenesis. It is necessary here, as he pointed out, to attempt to transfer the whole program of research to animals, as in the general field of experimental biology. Low,[3] Bloch[15] and recent authors[11, 16] have suggested the prophylactic use of patch tests in industry. Such a procedure may in time produce similar results. Normal workers may become sensitized by pre-employment patch testing. If we accept the definition of allergy[34] as a specifically altered state produced by a previous exposure and made manifest by subsequent ones, we are inviting such a state unnecessarily by making an exposure that may result in sensitization, which becomes evident upon employment. In rare instances there may be an occupational hazard which is considered irremovable; but even then the test is not dependable, for each patient has his own period of refractoriness.

Industry should test new chemicals upon guinea pigs, and thus find the sensitizing index of the chemical rather than that of the workers. It is easier to remove the hazard than the workers, and no chemical should be retained that, despite prophylactic measures, is known to be injurious to employees. Such chemicals can be eliminated, as has been shown by the excellent record made by the rubber industry in Massachusetts. Close supervision of workers is better than pre-employment patch testing.

The patch testing of normal individuals presents as many possible sources of error as does that of patients suffering from contact dermatitis, if not more. False positive reactions may result from too high a concentration at the time of testing, for the skin may vary in its response. Diet may influence sensitization in animals, as has been demonstrated by Mayer and Sulzberger.[35]

The application of a chemical under an occlusive dressing for twenty-four to seventy-two hours is not equivalent to an ordinary industrial exposure. The only similar exposure would exist where a worker spilled a solution on his clothes and failed to change them. False positives may cause unnecessary expenditure to eliminate suspected hazards, while the real hazard remains. They also create a spirit of distrust among employees who are unwilling to contact substances admitted to be hazardous to others; the result may be a walkout and the closing of the plant. False negatives, on the other hand, give a mistaken sense of security, and lull a suspicion which proper observation and investigation might confirm, for a single hypersensitive individual usually reacts in time to give warning of a hazard. In fact, an eruption of the skin may disclose a hazard of lethal properties, as shown by the recent work on chlorinated naphthalenes.[36] Powders and oils which interfere mechanically with bodily functions give negative reactions. How can one adequately test the dishwasher, the soda-fountain clerk, the cleaner of vegetables or the salad-maker whose hands are constantly wet for eight hours or more, or the woolworker, exposed to steaming solutions daily for years?

Furthermore, pre-employment patch testing is apt to make employes careless, supposing themselves immune—a mistaken notion, for Bloch[4] has shown conclusively that 100 per cent of subjects can be sensitized to an irritant. Routine patch testing of patients is to be deprecated; first of all, it is not practicable, for it is impossible to have sufficient test material on hand. The author has collected hundreds of samples of materials used in testing industrial workers, and these are rarely employed in routine patch testing. The white buck leather, for example, on hand may not in any way resemble the kind contacted by the patient, and the same holds true for powders, oils and solutions. Only one out of fifty oils may approximate the one contacted.

Sulzberger and Rostenberg,[37] after studying the results of over ten thousand tests made in a large clinic, stated that cutaneous tests were of little value for an etiologic diagnosis when applied in a disorganized or haphazard manner. They asserted that only those substances known to have been contacted or suspected as possible contacts should be employed. Twenty years ago, dermatologists thought scratch tests the solution of all their problems, but disappointed patients soon showed them their error. Today, patch tests are in vogue. They have proved most serviceable to the writer in industrial work, and are of greater value than they were a few years ago, as experience teaches better utilization. Yet it has been embarrassing to have a worker who has been assured after patch testing that there are no irritants in his occupation, return to the office after working a few hours, with a recurrence of his previous eruption; or to have a warning against a particular chemical prove of no avail because of other hazards which are present. Furthermore, relatives are apt to become inquisitive when a patient able to walk to the office is bedridden for days or weeks after a patch test. And difficulties are arising in cases of compensation and in medicolegal issues. A severe positive reaction to a known skin irritant, such as a liniment applied by a youthful colleague, makes considerable impression on a board member or a judge or jury, and one's explanations are likely to be dismissed as prejudiced.

Careful history-taking, knowledge of industrial exposures, personal investigations and the studying of industrial processes will reduce the necessity of routine patch tests. But when needed they will continue to be of distinct value in occupational dermatology. Wisely employed and interpreted, they will not suffer the fate of the scratch test, which has proved disappointing as an aid in dermatological research.

CONCLUSIONS

Patch tests have no diagnostic value. They have their fallacies and limitations, but with a carefully taken history showing definite exposure to the test material, and with a characteristic test eruption, a positive finding followed by the prevention of further exposures should result in a cure. Properly employed, the patch test is superior to both scratch and intradermal tests.

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REFERENCES

1. White, J. C.: *Dermatitis Venenata: An account of the action of external irritants upon the skin.* 216 pp. Boston: Cupples & Hurd, 1887.
2. Nestlar: quoted by Low.[3]
3. Low, R. C.: Skin-sensitiveness to non-bacterial proteins and toxins. Brit. J. Dermat. **36**:292-311, 1924.
4. Bloch, B.: The role of idiosyncrasy and allergy in dermatology. Arch. Dermat. & Syph. **19**:175-197, 1929.
5. Jadassohn: quoted by Sulzberger and Wise.[37]
6. Markley, A. J.: Anaphylactoid dermatitis due to direct contact with animal epidermal structures. Arch. Dermat. & Syph. **2**:722-724, 1920.
7. Biederman, J. B.: A new method of making multiple patch tests. Arch. Dermat. & Syph. **37**:198-200, 1938.
8. Reynolds, H. S.: A simple and efficient patch test. J. A. M. A. **104**:1925, 1935.
9. Biederman, J. B.: A case of contact dermatitis produced at a distance by the sensitizing agent. J. A. M. A. **106**:2236, 1936.
10. Wedroff, quoted by Urbach.[12]
11. Schwartz, L.: Sensitivity to external irritants in industry. New York State J. Med. **36**:1969-1976, 1936.
12. Shelmire, B., and Black, J. H.: A method of patch testing with plant oil. J. A. M. A. **108**:719, 1937.
13. Urbach, E.: Klinik und Therapie der allergischen Krankheiten. 746 pp. Vienna: Verlag von Wilhelm Maudrich, 1935.
14. Engelhardt, W., and Mahler, W.: Besteht eine Abhängigkeit des Ausfalls der Hautläppchenprobe nach Jadassohn-Bloch von Fieber und Schweiss? Dermat. Wchnschr. **99**:1205-1290, 1934. Abstract in Arch. Dermat. & Syph. **32**:298. 1935.
15. Bloch, B.: Occupational skin diseases from the biological point of view. J. State Med. **38**:373-382, 1930.
16. Sulzberger, M. B.: Industrial dermatoses. Indust. Med. **6**:594-597, 1937.
17. Sulzberger, M. B., and Wise, F.: The contact or patch test in dermatology: its uses, advantages and limitations. Arch. Dermat. & Syph. **23**:519-531, 1931.
18. Peck, S. M., and Salomon, G.: Eczema of infancy and childhood. I. Contacts as etiologic agents, with particular reference to feathers. Am. J. Dis. Child. **46**:1308-1328, 1933.
19. Peck, S. M.: Discussion on symposium on allergy. J. A. M. A. **103**:1292, 1934.
20. Obermayer, M. E.; Becker, S. W.; Praver, L. L.; Shaw, C., and Becker, F.: Epidermal and dermal hypersensitivity in patients with functional dermatoses as demonstrated by patch and scratch tests. Arch. Dermat. & Syph. **34**:411-420, 1936.
21. Brunsting, L. A., and Anderson, C. R.: Ragweed dermatitis: report based on 18 cases. J. A. M. A. **103**:1285-1290, 1934.
22. Sulzberger, M. B.: Acneform responses to patch tests: follicular and papulopustular reactions to selectively pilosebaccous irritants. Arch. Dermat. & Syph. **30**:566-569, 1934.
23. Ingram, J. T.: Dye dermatitis in relation to idiosyncrasy. Brit. J. Dermat. **44**:422-435, 1932.
24. Sulzberger, M. B., and Wise, F.: Drug eruptions. II. Dermatitis eczematosa due to drugs. Arch. Dermat. & Syph. **28**:461-474, 1933.
25. Spain, W. C., and Cooke, R. A.: Studies in specific hypersensitiveness. XXVII. Dermatitis venenata: observations upon the use of a modified extract from Toxicodendron radicans (L.). J. Immunol. **13**:93-112, 1927.
26. Straus, H. W.: Artificial sensitization of infants to poison ivy. J. Allergy **2**:137-144, 1931.
27. Field, H., and Sulzberger, M. B.: Experiments in poison ivy sensitivity. J. Allergy **7**:138-144, 1936.
28. Brown, A.; Milford, E. L., and Coca, A. F.: Studies in contact dermatitis. I. The nature and etiology of pollen dermatitis. J. Allergy **2**:301-309, 1931.
29. Schoch, A. G.: The patch test and the element of syringe contamination in arsphenamine sensitization dermatitis. J. A. M. A. **98**:1367-1372, 1932.
30. Cormia, F. E.: Experimental arsphenamine dermatitis. Canad. M. A. J. **34**:272-276, 1936.
31. Chargin, L.; Sulzberger, M. B., and Crowley, D. F.: Hypersensitiveness of the skin to arsphenamine as determined by contact test: its relationship to arsphenamine dermatitis. Arch. Dermat. & Syph. **22**:237-243, 1930.
32. Baer, H. L.: Lip-stick dermatitis. Arch. Dermat. & Syph. **32**:726-734, 1935.
33. Bloch, B., and Steiner-Wourlisch, A.: Die Sensibilisierung des Meerschweinchens gegen Primeln. Arch. f. Dermat. u. Syph. **162**:349-378, 1930.
34. Sulzberger, M. B.: Allergy in dermatology: a discussion of recent contributions and of their relationship to earlier dermato-immunologic studies. J. Allergy **7**:385-427, 1936.
35. Mayer, R. L., and Sulzberger, M. B.: Zur Frage der jahreszeitlichen Schwankungen der Krankheiten. Der Einfluss der Kost auf experimentelle Sensibilisierungen. Arch. f. Dermat. u. Syph. **163**:245-262, 1931.
36. Report of the Division of Occupational Hygiene. Massachusetts Department of Labor and Industry, 1936.
37. Sulzberger, M. B., and Rostenberg, A.: Some results of patch tests. Arch. Dermat. & Syph. **35**:433-454, 1937.

DISCUSSION

DR. MILDRED L. RYAN, Brockton: Dr. Downing has covered the subject in such a comprehensive and satisfactory manner that there is very little more to say. I agree with him that the function of patch tests is largely corroborative and of little value if applied haphazardly. It is a measure by which one may eliminate some of the suspected etiologic agents in contact dermatitis rather than a valuable means of diagnosis. A careful history is most important.

In my experience, the patch test has been of most value in cases of occupational dermatitis. If a patient is tested with several irritants to which he has been exposed at work, and is found to have a positive reaction to only one of them, he may often be able to continue his job if he does not come in contact with this substance. This is very important from the point of view of the patient as well as that of the insurance company. To cite a case: A man who worked as a spreader in a rubber factory handled several types of stock. He had two attacks of acute dermatitis within a month, the second appearing six days after he returned to work. The first cleared after two weeks, the second after a month. A patch test with No. 800 stock gave the only positive reaction. He returned to work and did not come in contact with this material for three months and his skin remained clear. One day a co-worker tipped over a tub containing No. 800 stock and the patient assisted him in lifting the tub. He came in contact with the rubber for less than five minutes. The next day he had an acute dermatitis of the hands which lasted two weeks. This case shows that the skin, once it has become sensitized to a given irritant, usually reacts very quickly to the substance on repeated exposures.

In evaluating the importance of patch tests I think it is of interest to mention that Rostenberg and Sulzberger* found that 17.8 per cent of the patients with a contact dermatitis produced by topical remedies showed a positive reaction when patch-tested with 5 per cent ammoniated mercury. They also found that tests with ammoniated mercury yielded a higher percentage of positive reactions than did tests with any other substance on their list of materials that were employed as external medicaments. This is rather important since ammoniated mercury is used so frequently with and without the advice of physicians.

A negative reaction to a patch test does not always mean that the substance tested was not the cause of the

*Rostenberg, A., Jr., and Sulzberger, M. B.: Some results of patch tests: a compilation and discussion of cutaneous reactions to about five hundred different substances, as elicited by over ten thousand tests in approximately one thousand patients. Arch. Dermat. & Syph. **35**:433-454, 1937.

eruption. Spontaneous desensitization may occur. Furthermore, patients may be sensitive to the substance in question in the skin areas in which the dermatitis appears, and not sensitive in the areas tested. This has been shown by Sulzberger and Goodman* in a case of cheilitis due to lipstick in which patch tests with the causal lipstick gave repeatedly negative results, while subsequent patch tests with undiluted dyes showed many strangely positive reactions. This was, no doubt, due to the fact that the sensitivity of the skin was less than that of the lips.

I believe in applying patch tests only when indicated and not as a routine pre-employment measure which some dermatologists favor.

DR. BERNARD APPEL, Boston: This has been a very comprehensive presentation of an important subject, and many significant and valuable observations were made by Dr. Downing. It might not be too elementary to draw attention again to the relation of the type of test to the reaction of the skin in the determination of a suspected agent. The intradermal and scratch tests are obviously not indicated as diagnostic procedures in cases of dermatitis due to external irritants acting by contact with the skin. The patch test is the only test which has here any connotation of specificity.

But as soon as we bring into the discussion any interpretation of a specific effect, we become at once involved in a controversy. Is this particular person peculiarly susceptible to the effect of this special substance on his skin? or, Is the action of this special substance one of primary skin damage? In other words, Is the effect on the skin produced by antigen-antibody interaction or by direct chemical action? If by direct chemical action, the test loses a great deal of its specificity because the chief role is played by the offending substance itself, and the effect is the resultant of forces outside the skin, such as concentration of substance, temperature at which reaction takes place, duration of exposure, and so forth. If, however, the effect on the skin is specific, that is, allergic, then the skin has, by commonly accepted definition, reacted differently to recent concentrations of the offending substance than it did to the original exposures which sensitized it. But to differentiate these two mechanisms by a patch test requires more than the mere performance of the test. Would not the application of cantharides produce a positive patch test in a person with dermatitis caused by potassium bichromate? For example, a maintenance engineer developed a dermatitis on his hands. He was tested with kerosene, swabbed over an area of several square inches, which was then covered with gauze and adhesive plaster. The resultant reaction after twenty-four hours can well be imagined, but the conclusion that his dermatitis was caused by contact with kerosene on the basis of this test can with equal obviousness be seriously questioned. May not a positive test be a symptom of the condition of the skin rather than an index of the cause?

The different skin areas vary in their reactivity to the same substance; a negative patch test on the forearm does not exonerate a substance suspected of causing a dermatitis on the face. It may sometimes be necessary to test the identical site of the recently healed dermatitis with a suspected substance. Such a procedure is occasionally dangerous. Often the site of a previous dermatitis may have become increased in sensitivity to a different agent than the original offender.

Related compounds may produce mutually increased sensitivity. For instance, the application of a lotion containing phenol in the treatment of dermatitis caused by toluol may produce a disastrous effect.

It is therefore significantly evident that the correct interpretation of the results of a patch test requires experience, special study of the situation involved, knowledge of the reactivity of the skin and expert evaluation of all the conditions surrounding the procedure.

Mention has been made of ammoniated mercury ointment. It is my personal opinion that sensitivity to ammoniated mercury, and to other similar compounds, may rest in some cases on the basis of specific allergic sensitivity, and in other cases on that of specific chemical damage. As a result of this opinion, it has been my custom, if I use ammoniated mercury ointment in the treatment of infections such as impetigo, to prescribe a 10 per cent preparation, because if patients are going to react to that percentage, they will almost always, and with an approximately equal degree of severity, react to whatever other percentage happens to be prescribed, whether 5 per cent, 2½ per cent or lower.

I am compelled to agree with Dr. Downing and Dr. Ryan that the routine, indiscriminate use of patch testing with large numbers of generally recognized irritants is to be deprecated. Rather, a very careful and complete clinical analysis should be made of the individual case and only properly indicated substances should be used as test materials.

*Sulzberger, M. B., and Goodman, J.: Acquired specific hypersensitivity to simple chemicals. II. Cheilitis, with special reference to sensitivity to lipstick. Arch. Dermat. & Syph. 37:597-615, 1938.

THE USE OF THE SCRATCH TEST IN DERMATOLOGY*

With Special Reference To Atopic Dermatitis

JOSEPH GOODMAN, M.D.†

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AT THE present time many physicians who are not primarily allergists are equipped to perform scratch tests on allergic patients. That these tests are in many cases productive of valuable information has been demonstrated time and again, and needs no emphasis here. Of greater concern is the fact that except in patients with hay fever and in certain patients with asthma, the general practitioner as well as the specialist is not infrequently dissatisfied with the practical results which he derives from performing scratch tests. This is particularly true with reference to patients with diseases of the skin.

It is not within the scope of this paper to discuss the technic of preparing extracts for testing, of performing and reading the scratch test and of using the intradermal form of testing. The purpose of this discussion is rather to define the proper use of the scratch-test procedure in dermatology. For like every other laboratory procedure, it is pertinent in certain cases and superfluous in others. Only by knowing when to test the patient and when not, can satisfactory results be obtained.

The first and essential step, then, is the correct selection of the patient to be tested. To choose intelligently the physician must first of all be able to establish a dermatological diagnosis. It will serve only to discourage both the patient and the physician if, for example, these tests are performed on individuals suffering from psoriasis, seborrheic dermatitis, lichen planus or alopecia areata. Thus far it has been impossible to demonstrate any significant allergies in patients with these and many other skin diseases.

In this connection it must be remembered that even a certain percentage of apparently normal individuals give positive reactions to scratch tests—and particularly to intradermal tests.[1] The occasional finding of positive tests in patients with these skin diseases cannot, therefore, be considered significant, since in the first place the incidence of positive tests is no greater than in normal persons, and in the second place, elimination from the diet or the environment of the substances which produced the positive reactions does not help the patient.

Not only is this true of diseases which so far as our present knowledge goes are not based on allergy, but it is also true of certain common skin diseases which are primarily allergic in nature. First of all, contact dermatitis, which is most often based upon a specific acquired hypersensitivity and is therefore usually an allergic disease, must be investigated by means of the patch test, which is read twenty-four to forty-eight hours after the application of the test substance.[2] Secondly, the drug eruptions, which for the most part are also based upon specific acquired hypersensitivity, cannot as a rule be investigated by means of scratch tests. In fact, skin tests of any sort are on the whole disappointing when applied in cases of drug eruption. In a third group of diseases the test must be intradermal, and must be read forty-eight hours after application. The tuberculin test, the Frei test for lymphogranuloma inguinale and the intradermal test with fungous extracts such as trichophytin and oidiomycin are examples of this group.

These varieties of allergy as well as the fourth variety which should be investigated by the scratch test, differ from one another in the mechanism of the reaction to testing. In spite of the fact that, as generally recognized since the time of von Pirquet and as again emphasized particularly by Landsteiner,[3] there is some evidence of a fundamental relation between the various manifestations of allergy, it is nonetheless true that there are obvious differences between the different forms, and that the practical procedures of investigation differ for the various categories of allergic skin diseases.

The dermatoses which should be investigated by the scratch test are those which are atopic. "Atopy" is a word coined by Coca and Cooke[4] for allergic diseases which have certain characteristics in common. It is derived from a Greek word which means "strange." These diseases are strange or unusual, for example, in that they are accompanied by a particular variety of circulating antibody which so far as we know is peculiar to man. It is known as the Prausnitz-Küstner body, passive-transference antibody or reagin. This is a substance which circulates in the blood

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stream of patients who are subject to the atopic diseases. Serum from these patients injected into the skin of normal individuals has the property of passively sensitizing the injection site. For example, if serum from a patient who presents a positive scratch test to egg white is injected into the skin of the forearm of a patient who is not skin-sensitive to egg white, a subsequent scratch test with a water-soluble extract of egg white performed at the site of injection will in many cases elicit a positive wheal reaction. A similar site into which sterile saline has been injected and a third untreated site which is employed as a control will at the same time show no reaction to a scratch test with the same egg-white extract.

The passive-transference antibody is demonstrated by the immediate wheal reaction, a reaction which reaches its maximum twenty to thirty minutes after the application of the test substance to the previously sensitized skin site. It is essential to give the extract (or allergen) access to the blood vessels of the skin, for it is probably an alteration of the permeability of the walls of these vessels that produces the positive scratch-test reaction.

The presence of the immediate wheal reaction produced without previous passive sensitization of the skin is a second characteristic of patients with atopic disease. Further characteristics are a family history of similar diseases, a past history of other atopic diseases and the presence of eosinophilia, both locally at the site of the scratch-test reaction and in the blood stream. These five characteristics are found quite regularly but not invariably in atopic individuals.

The most important atopic diseases are hay fever, certain varieties of asthma and vasomotor rhinitis, and finally a dermatosis which is best designated as atopic dermatitis,[5] and under which are grouped many of the cases of infantile eczema. In the investigation of atopic dermatitis the dermatologist finds the most important use for the scratch test.

Atopic dermatitis is a disease which has many names. Among them are generalized neurodermatitis, flexural eczema, prurigo with lichenification and prurigo diathèsique of Besnier, to mention only a few. For several reasons the name atopic dermatitis was chosen by Sulzberger and Coca[5] to replace these older names. First, it emphasizes the primary importance of the multiple atopic hypersensitivities which are found in the great majority of patients with this disease. Secondly, it avoids the use of the word "eczema" by substituting therefor the word "dermatitis." This disease is not eczema, for it presents few of the clinical and none of the histological characteristics of true eczema. Of particular importance in this respect is the absence of vesiculation, unless it is induced by improper treatment or by a superimposed contact dermatitis. Thirdly, this new name, unlike the name neurodermatitis, does not suggest that the nervous system plays a role of primary importance in the production of this dermatosis. In fairness it must be pointed out that certain dermatologists, notably Obermayer, Becker, Praver, Shaw and Becker,[6] do not concede that allergy plays the most important role in the pathogenesis of this disease. However, its significance has now been recognized by many dermatologists and by most allergists, and nervous influences are generally believed to play a subsidiary part.[7]

Atopic dermatitis occurs for the most part in families in which other atopic diseases appear. An excellent description of the course of the disease has been given by Hill and Sulzberger.[8] In summary it may be said that it is a chronic, relapsing and pruriginous dermatosis which can be divided into three episodes. The first episode is infantile eczema. The atopic infantile eczema often cannot be distinguished by its appearance from the seborrheic or contact varieties. Be this as it may, the incidence of infantile eczema in the past history of young adults with atopic dermatitis is exceptionally high. Usually, but not always, after an interval of freedom from skin disease and between the ages of two and twelve the second episode occurs. At this time the dermatitis shows a definite tendency to localize in the antecubital and popliteal areas. The third episode, again, usually follows an interval of freedom from dermatitis, has its onset from the age of puberty to early adult life and is again primarily flexural in distribution, although the face and neck are also commonly involved. Occasionally in this period the eruption is extensive, involving the scalp, chest, backs of the hands, abdomen, back and buttocks. Any one or two of these episodes may be missing. The disease is quite rare after the age of thirty.

The investigation of patients with atopic dermatitis reveals that during the first episode the principal hypersensitivity is to foods. Since the diet of the infant is limited, tests with about a dozen foods usually suffice to uncover the offenders, the worst of which are, ordinarily, wheat, milk and eggs. In the light of the test results the physician can devise elimination diets which have a fair prospect of benefiting the patient. However, local treatment should not be neglected.

During the later course of the disease the hypersensitivity to foods may be lost but multiple new hypersensitivities appear. These are mostly directed toward inhalant substances, such as animal danders, feathers, pollens and especially silk. The

identification of the substances to which the patient is hypersensitive and the elimination of these allergens from his diet or his environment will in some cases do much to make the patient more comfortable. This is especially true when elimination is combined with the proper local treatment of the skin. It must be admitted, however, that elimination rarely if ever results in a cure. This may be because it is apparently impossible to discover and eliminate all the allergens to which the patient is hypersensitive. However, this should not deter the physician from making a thorough investigation and attempting as much elimination as possible. Unfortunately, desensitization procedures have not as yet been successful in the treatment of patients with atopic dermatitis.

At this point and in the light of the foregoing discussion it is necessary to consider for a moment the significance of the wheal reaction to the scratch test. It has been known for some years and has recently been re-emphasized by Baldwin and Glaser[9] that the size of the wheal reaction shows no constant change during desensitization procedures for asthma or hay fever. Further, it is well known that once a positive scratch-test reaction is present it may persist for years, even long after clinical sensitivity to the test substance has been lost. For example, a positive reaction to ragweed pollen may persist long after the patient has ceased to have hay fever. Therefore in atopic dermatitis, as well as in all other atopic diseases, scratch tests must be used only as guides to elimination. A positive scratch-test reaction does not necessarily mean clinical sensitivity to the test substance. Conversely, there are certain cases in which clinical hypersensitivity to a substance is not accompanied by a positive scratch-test reaction. Under such circumstances the significance of a given allergen can be proved only by its elimination, with subsequent re-exposure of the patient. It follows that the results of the scratch-test investigation, like those of all other laboratory procedures, must not be accepted blindly.

Thus far the discussion has been confined to the use of the scratch test as a guide for the treatment of atopic dermatitis. Occasionally it is of advantage in diagnosis to differentiate this disease and seborrheic dermatitis or contact dermatitis. In obscure cases the presence or absence of scratch-test reactions may be of considerable diagnostic significance.[10]

Apart from its employment in atopic dermatitis, the scratch test is of limited value in dermatology. Some authors report that in patients with resistant acneform eruptions — probably not acne vulgaris — food sensitivities may be revealed by scratch tests followed by elimination procedures.[11] In the experience of most dermatologists such cases are rare. Occasional patients with dermatitis herpetiformis present positive scratch-test reactions which are of doubtful significance. It is perfectly reasonable also to use the scratch test in the investigation of certain dermatoses of obscure etiology but only after all the customary means of establishing a dermatologic diagnosis have failed. For the most part, the physician who resorts to scratch tests in such cases must expect to be disappointed in his results.

The discussion of acute and chronic urticaria and angioneurotic edema has been reserved for the last in order to emphasize the limited value of the scratch test in the investigation of these diseases. Only about half the patients with urticaria present positive reactions to scratch tests, and very few of these benefit by the elimination of the substances which produce the reactions.[12] Indeed, there is as yet no proof that the majority of urticarias are allergic in origin. Scratch tests are indicated for patients with chronic urticaria if they have a family or personal history of atopic diseases, or if the usual treatment by the elimination of drugs, obvious foci of infection, and so forth has failed. In the ordinary case of acute urticaria, scratch tests are superfluous, for these patients generally effect a speedy recovery.

One exception to the usually fruitless investigation of urticaria by the scratch test may be noted. In the last few years patients who develop urticaria and generalized reactions from the injection of organ products such as insulin,[13] pituitary extract[14] and liver extract[15] have been shown to present wheal reactions to scratch tests with the offending substances, as well as specific passive-transference antibodies. These findings are of considerable theoretical importance, since it has been shown in each of these cases that the hypersensitivity is directed not toward a substance peculiar to the animal from which the extract was derived (species specific), but toward a substance peculiar to the organ and present in that organ in many different mammals (organ specific). At present, however, this finding is not of extreme importance to the practitioner, since such cases are quite rare.

In general, the practitioner will do well to lessen his use of the scratch test in the investigation of dermatological diseases, and to apply the patch test more freely. The employment of the scratch test for patients with infantile eczema and atopic dermatitis is certainly justified when indicated, and should not be neglected. However, in the investigation and treatment of other dermatoses the scratch test is in most cases of no value.

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REFERENCES

1. Backemann, F. M., and Simon, F. A.: Technic on intracutaneous tests and results of routine tests in normal persons. J. Allergy 6:184-188, 1935.
   Grow, M. H., and Herman, N. B.: Intracutaneous tests in normal individuals; an analysis of 150 subjects. J. Allergy 7:108-111, 1936.
2. Sulzberger, M. B., and Wise, F.: Drug eruptions. II. Dermatitis exfoliativa due to drugs. Arch. Dermat. & Syph. 28:461-474, 1933.
3. Landsteiner, K.: Serological and allergic reactions with simple chemical compounds. New Eng. J. Med. 215:1199-1204, 1936.
4. Coca, A. F., and Cooke, R. A.: On the classification of the phenomena of hypersensitiveness. J. Immunol. 8:163-182, 1923.
5. Coca, A. F.: Specific diagnosis and treatment of allergic diseases of the skin; present status. J. A. M. A. 103:1275-1277, 1934.
6. Obermayer, M. E.; Becker, S. W.; Praver, L. L.; Shaw, C., and Becker, F.: Epidermal and dermal hypersensitivity in patients with functional dermatoses as demonstrated by patch and scratch tests. Arch. Dermat. & Syph. 34:411-420, 1936.
7. Sulzberger, M. B., and Goodman, J.: Relative importance of specific skin hypersensitivity in adult atopic dermatitis. J. A. M. A. 106:1000-1003, 1936.
8. Hill, L. W., and Sulzberger, M. B.: Evolution of atopic dermatitis. Arch. Dermat. & Syph. 32:451-463, 1935.
9. Baldwin, L. B., and Glaser, J.: Effect of treatment on skin and mucous membrane sensitivity and on reagins in hay fever. J. Allergy 8:129-142, 1937.
10. Sulzberger, M. B., and Rostenberg, A., Jr.: Practical procedures in the investigation of certain allergic dermatoses. J. Allergy 6:448-459, 1935.
11. White, C.: Acneform eruptions of the face; etiologic importance of specific foods. J. A. M. A. 103:1277-1279, 1934.
12. Stokes, J. H.; Kulchar, G. V., and Pillsbury, D. M.: Effect on the skin of emotional and nervous states: etiologic background of urticaria with special reference to the psychoneurogenous factor. Arch. Dermat. & Syph. 31:470-499, 1935.
13. Tuft, L.: Insulin hypersensitiveness: immunologic considerations and case reports. Am. J. M. Sc. 176:707-720, 1928.
14. Simon, F. A.: Hypersensitiveness to pituitary extract. J. A. M. A. 104:996, 1935.
    Simon, F. A., and Ryder, C. F.: Hypersensitiveness to pituitary extracts. J. A. M. A. 106:512-515, 1936.
15. Criep, L. H.: Allergy to liver extract. J. A. M. A. 110:506-508, 1938.

Discussion

Dr. C. Guy Lane, Boston: I wish to emphasize three points in the discussion of Dr. Goodman's paper. It may seem elementary, in the first place, to lay emphasis on the need for correct diagnosis. And even after a diagnosis of dermatitis or eczema has been made there is further need of refinement of this diagnosis in indicating as far as possible whether the condition is seborrheic dermatitis, industrial dermatitis, contact dermatitis or atopic dermatitis. Dr. Goodman has reviewed the five striking characteristics of this condition. He has cited the three types of episode which occur, and has made an excellent summary of this entity, which deserves much wider attention than it has had in the past.

The second point I desire to emphasize is the diagnosis of the type in which there is a reasonable hope of achieving success with our skin tests, as well as the decision as to which type of skin test will be best — whether to use the patch test, the scratch test or the intracutaneous test. This point has been emphasized by previous speakers but needs further repetition. There is much indiscriminate testing in which there is too little chance of achieving helpful results.

The third point I wish to make relates to interpretation. Albumin in the urine does not necessarily mean nephritis, any more than sugar means diabetes; nor does a positive Wassermann test absolutely mean syphilis. Patients having skin tests done and physicians sending patients for consultation frequently ask first regarding the report of the skin tests. Such emphasis on these tests is not warranted. They cannot be considered as conclusive unless supplemented by other findings, for positive tests are found without supporting clinical evidence. The reverse is also true.

Brunsting* in his excellent discussion of atopic dermatitis in 1936 called attention to the discrepancies found in comparing skin tests and the clinical evidence and symptoms which are presented. We should not, at least as yet, place too much dependence on skin tests, but rather should conduct a careful study of the individual case, particularly with regard to the history. History-taking perhaps is not the lost art that it was formerly thought to be. Environmental factors, psychological or psychiatric factors and careful elimination studies need consideration. The data from such studies will give us the best answer today with the rational use of skin tests.

*Brunsting, L. A.: Atopic dermatitis (disseminated neurodermatitis) of young adults: analysis of precipitating factors in one hundred and one cases and report of ten cases with associated juvenile cataract. Arch. Dermat. & Syph. 34:935-957, 1936.

Dr. Francis M. Thurmon, Boston: I congratulate Dr. Goodman on the excellence of his paper. It is so filled with significant statements which are easily and rapidly read, that, like psychiatry, unless we know our subject well and dig for that which we are searching, there is danger of passing over much that is worth while.

Certainly there is truth to the statement regarding the selection of dermatologic cases for scratch testing. Infantile eczema, atopic dermatitis, seborrheic dermatitis and contact dermatitis in their differentiation from atopic dermatitis, and certain other dermatoses where the usual diagnostic procedures have been unsuccessful and cutaneous allergy of the vascular and epithelial type is a possibility, form the chief entities for scratch testing.

We must keep in mind that the skin is a highly complex organ capable of many responses. There are fifteen or more different types of response that we already know. The response in atopic dermatitis is one of them.

Infantile eczema and atopic dermatitis are usually hereditary. The allergens are mostly proteins or are water soluble. In atopic dermatitis the skin testing is by scratch or injection, also by ingestion. There are strains of sensitivity that run through these families. The shock tissue is the skin. These people have a constitutional irritability; they seem to come from hypersensitive stock.

In infantile eczema there is usually a natural desensitization — they outgrow it for a time. Then later they may show multiple allergy, such as hay fever and cutaneous sensitivity in the same individual. They may react to ragweed in season; puberty influences the reactivity, the menopause likewise; and other factors such as heat, and light are reflected in the skin manifestations of the reactive types.

In conclusion it may be stated that these allergic atopic individuals with their complex sensitivity form a puzzling group. The skin tests in the final analysis are not conclusive unless they are substantiated by other factors, namely the history and the further clinical course of the case.

I should like to ask Dr. Goodman one question, What is his opinion of the indentation method of skin testing as compared with the scratch test?

Dr. Joseph Muller, Worcester: I have been using skin tests for a longer time than most men here, and I get some results from them, but I cannot agree entirely with what has been said. The paper was excellent, but it was too short. Dr. Goodman did not emphasize a good many things that, to my mind, are important. The diagnosis of atopic dermatitis does not mean that we have to apply scratch tests immediately; on the contrary, if you take a careful history you will be able to point to the offending food much faster than if you make tests.

Another point is that if skin tests are made they must be done by those who know how. Skin tests are usually made by laboratory technicians, — at least they are in Worcester, — who have not the slightest idea of how to do them, for they were taught by pathologists who never

did any work in this field and do not know much about skin testing themselves. Even tests done by experts are only 70 per cent right, and it is obvious that unreliable tests are useless. Quite often, instead of skin-testing patients I am using elimination diets, as recommended by Rowe, of California, with a great deal better results than those following scratch tests. Of course we occasionally get spectacular results from just a few scratch tests.

Another thing to remember is the acneform dermatitis which was first described by Cleveland White, of Chicago. In most cases we see an eruption on the face and back resembling acne, but the patient is past adolescence and the usual treatment for acne does not help. Elimination diets or skin tests and the excellent results obtained from them will prove that we have been dealing with an atopic dermatitis rather than acne.

A PHYSICIAN: I should like to add one more point in regard to scratch tests. They can show past sensitization or present sensitization, but they can also show oncoming sensitization. This has been more or less proved by Colmes, Guild and Rackemann,* who tested bakers who had been in the business anywhere from five to twenty years. None of them gave a history of sensitization, either personal or familial, and yet about 40 per cent gave positive scratch-test reactions to one or more of four substances with which they were tested.

DR. GOODMAN (closing): I thank those who discussed this paper for bringing out points which can certainly stand emphasis. I am quite in agreement with Dr. Muller that there are items which could be elaborated on. However, for the sake of conciseness this elaboration has been omitted.

With regard to Dr. Thurmon's question, I have had no experience in doing scratch tests by the indentation method. I have done them by making a scratch with a needle through the skin as superficially as possible, enough to break the epidermis but not to go into the cutis, and also by means of a small instrument which produces a round incision of constant proportions in the epidermis.

Dr. Muller mentioned two points on which I should like to comment very briefly. First he mentioned the work of Rowe on food allergy. As Dr. Muller says and as Rowe and others have amply demonstrated, the results of scratch tests with food extracts do not always agree with the clinical manifestations of food allergy. But the treatment of food allergy by elimination is often very difficult, especially in general practice.

Secondly, Dr. Muller mentioned the importance of foods in acne vulgaris. It is common experience that some foods will always make certain acne patients worse. Scratch tests for these foods are usually negative. However, there are certain patients with acneform eruptions (probably not acne vulgaris) whose lesions start as papules, itching being a prominent symptom. For such patients I believe that scratch tests are of value in disclosing possible causes of the skin lesions.

*Colmes, A.; Guild, B. T., and Rackemann, F. M.: Studies in sensitization: influence of occupation on sensitization in man as determined in a study of thirty-two bakers. J. Allergy 6:539-546, 1935.

SYPHILITIC SCARS OF THE SPIRIT*

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TWO recent conversations in regard to syphilis have jarred me into a desire to register a protest against the idea of confining a syphilitic patient's treatment to just what can be pressed through a needle into a vein or a buttock.

The first of these conversations was with a patient who had been entirely without treatment for three months after the diagnosis of secondary syphilis was given to her. This delay was caused by a remark passed by the doctor whom she first consulted. According to her story, which seems to be a true one, when the doctor told her that her diagnosis was secondary syphilis she asked how much treatment would be required. The doctor told her that two years of weekly visits would be necessary. She then asked how much this would cost and was told that there were fifty-two weeks in a year; if she multiplied that by two and then by ten dollars she would arrive at the correct sum. She replied that she could not possibly afford to pay for it; to which the doctor retorted, "Go out on the street and earn the money the same way you got the disease." The brutality of this remark led her to leave his office without even the initial treatment and then wait three months before summoning up enough courage to go to another doctor, by which time her rash was extremely disfiguring. The doctor who had been so brutal had not even bothered to learn the cause of her infection, which seemed definitely to have been a wedding present from her husband.

The second of these conversations was with a health officer, fortunately in a far-away state, who told me that the only method he used to keep patients under treatment was to "crack the whip," as there was no sense in trying to persuade them. When I asked him if he frequently "cracked the whip" he proudly stated that he did.

There is as we all know a great similarity in the problems of gonorrhea and of syphilis, from the public-health point of view. Yet men with gonorrhea as a rule seem to be relatively little disturbed, perhaps believing that a man is not grown up until he has had it; on the other hand,

*Read at the annual meeting of the Massachusetts Medical Society, Boston, May 31, 1938.

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it seems that comparatively few, except the most ignorant and callous, fail to be upset over having syphilis. Very commonly the mental anguish they go through during the first few hours, usually weeks and commonly even months, is a problem at least as perplexing as that of the drugs to be given, and at times far more so.

It should be obvious to all who have anything to do with gonorrhea or syphilis that by all odds the most important contact between the patient and the physician is the first one. Frequently the entire success in the handling of the problem is determined by the first visit. On that may depend the success of a life, or a failure leading to general paresis, or a congenital syphilitic infant. In a busy practitioner's office, regardless of the dozen patients sitting in the waiting-room, the one with suspected syphilis must be considered that day's emergency. The physician's failure to meet this emergency successfully may, years later, cost the patient his life.

At this first visit one must explain in simple, straightforward language what is ahead of the patient: that the recurrences, disappointments, chances of infection of the mate, danger of having congenital syphilitic children and serious or even tragic late complications, such as actually used to occur not rarely under the old mercury regime, can all be controlled now, provided adequate treatment is given and the patient does his part by co-operating and taking treatment properly. Even the seemingly worst situations, such as a young couple with early syphilis and the girl pregnant, need cause but little alarm, for if the case is properly handled the probability is that the parents will be cured and the fetus saved.

To nearly all patients syphilis means venereal disease. Of course we all really knew even before Nelson gave us the term "genitoinfectious disease" that syphilis is usually not a venereal disease. There is first the congenital type, and the most extreme cannot consider that as proof of immorality. Next there is the accidental type, and whether this is incurred by the physician in line of duty or by a young woman who kisses her fiancé, it is obviously not venereal. Occasionally a wife gives it to her husband, and far too commonly the husband gives it to his wife. In neither case can the recipient fairly be accused of having venereal disease. We in the medical profession have always known that, but do we take time enough to clear up that point with our patient? Are we sure that we ourselves are not guilty of using this expression without cause?

Even after seventeen years I have failed to help one patient understand that her disease was not venereal. In spite of my having been instrumental in the girl's keeping her job as a food-handler, and later taking her into my own family as nursemaid; in spite of my giving her frequent long talks during the active treatment and every time she came into the office for yearly check-ups, she still persists in thinking herself a moral leper because of a primary lesion on her lip, acquired from a man to whom she was engaged. The case is unusual only in that it is an extreme one.

Of course, in a situation where the moral issue is present this must be met frankly and squarely, and above all, with understanding. Here is where careless or indifferent handling may so antagonize the patient that he will refuse all co-operation; but usually, if by interest and understanding he can be made to feel that every question is put with the definite purpose of finding out something of value to him, or is asked from a public-health point of view so that the knowledge thus gained may help to prevent a similar mishap to others, he will tell everything freely.

Some idea of the upset can often be gained the first few minutes after the diagnosis has been given, when a considerable number of patients immediately threaten or question the advisability of suicide. So far I have known only two who have immediately carried out their threats. I was not acquainted with one of them, but I knew how painstakingly the doctor had explained the case to him, and the doctor was one of the kindliest and most understanding men it has ever been my privilege to know. As the man left the office he thanked the doctor for all he had done for him, but remarked that he would go only halfway across the bridge over the river — and he did just that.

The other patient was mine, and I shall never know what error I made. He said that he had had gonorrhea three or four times and that his wife had never been unkind about it, so I was entirely unaware that he was disturbed. The headlines in the following morning's paper told me of my mistake, but I still do not know how I failed him.

Another patient, a girl in her sixteenth year, met a traveling salesman. He asked her to marry him when he was next in town. This happened fifteen years ago and she has never laid eyes on him since, but while his image was very vivid in her memory, spirochetes were found in a lesion on her lip. A bad blunder was made, for no one thought to straighten out the thoughts of injustice going through the girl's mind: "If she had picked up syphilis by merely kissing a man, what was the use of behaving anyway?" One who understands human nature would be pretty callous if he could not understand this girl's logic and sym-

pathize with it. So when she whispered to us two months later that besides her injections we should give her an examination for pregnancy, we realized the mistake we had made, especially when we also found gonorrhea. During the next two months, while arrangements were being made for some financial help from the man responsible for her pregnancy, she learned that this was far from a casual affair on his part and that he loved her deeply. The resentment she had felt toward him was soon lost, and in the years since then she has been very happy, with a kind husband and three lovely children.

I cannot refrain from offering the two following cases to illustrate the difference in reaction of two patients to a diagnosis of syphilis.

A very highstrung but likable boy had been coming to the hospital clinic for several years for treatment of congenital syphilis. When he became a college freshman he happened by accident to see the diagnosis on his record. He said nothing to anyone of his discovery, nor did he give a hint that he was upset by it, but went immediately to a ticket office of one of the air lines and bought a ticket home to a Far-Western state. Very fortunately he talked in his sleep. His room-mate heard him and found out what the boy planned to do, namely to go home and kill his father. The room-mate tried to argue him out of it, and when this did not work tried to get the air line to cancel the passage. They refused, thinking it was some college prank. There was only one course left for the room-mate, and he took it. Both boys went to the airport; there the room-mate staged such a row that the pair were arrested, and naturally the passage was canceled. Later an understanding doctor took this boy in hand and explained the entire case to him so carefully that he was able to realize just how hotheaded and foolish his previous attitude had been.

The second case was that of a woman of a type not usually found. When the diagnosis was given her she broke down completely. Naturally the first thought was that she was upset because of her own condition. Not at all. The only thought in her mind was her attitude toward a neighbor suspected of suffering from the same disease. She felt she had been heartless, unsympathetic and utterly disagreeable in her treatment of that neighbor.

In the first case the reaction shows nothing but resentment and hatred toward the suspected cause of the infection; the second only unhappiness because of failure to show sympathy and understanding toward an unfortunate neighbor.

I do not know of any emotional upset occurring as regularly as the suffering of a parent who realizes that the interstitial keratitis of a promising son or an adorable daughter is due to a foolish early misstep on his or her part.

It is very unfortunate that there has been a persistent delusion in the minds of many people regarding the infectiousness of this disease. A great many still believe that it is so terrible that not only must marriage be forbidden but even the common contacts of life should be avoided. I have in mind one very popular middle-aged woman who developed a gumma on the wrist. The very day the diagnosis was given she resigned from all her women's clubs and church activities and decided to be an absolute recluse for the rest of her life. Fortunately from a chance remark this was discovered before it was too late for her to rejoin, but more than six months passed before she summoned up enough courage to resume her attendance.

Years ago a young woman came to the clinic with secondary syphilis, stating that there was a history of just one contact, her fiancé. Examination of the fiancé revealed an accidental syphilis with a trivial primary lesion of the lip and a rash so faint that its significance had never dawned on him. Both man and woman remained under treatment and subsequent semiannual examination for six or seven years. When the woman returned for one of the check-ups it was obvious that she had lost a good deal of weight, in fact 25 pounds. Painstaking examination failed to find any cause. Finally some chance remark by one of the doctors in the clinic whom she felt she could trust drew out the story. Her fiancé was anxious to give her a good home, but she had heard so many remarks by patients waiting in the clinic for treatment to the effect that it was never safe for syphilitics to marry that she had not dared ask the doctors for fear they would confirm them. The couple were encouraged to marry at once. It was wonderful to see the change in her on a six-months check-up. Her normal weight had returned and there was real happiness in her expression, added to which she looked ten years younger. I believe we had dismally failed on our first contact with that patient.

There seems to be a woeful ignorance on the part of many as to the precautions necessary. In fact, one patient recently asked how long before it would be safe to stop boiling his dishes. This was in 1937, and his syphilis had been acquired during the Spanish-American War. During all these years he had boiled his dishes and even his underwear. At the time of his infection *Treponema pallidum* had not been discovered, the limits of infectious periods were not well understood, and treatment did little toward controlling

infectiousness. We know now; yet no one had thought to explain to this man that in the usual case infectiousness through ordinary contacts dies out in a year or two in both sexes, and that under treatment the infectious period is usually over in much less than a month; in fact, dark-field examinations of primary lesions indicate that it is ordinarily passed within forty-eight hours after the first injection.

A known syphilitic under treatment is of no possible danger to his fellow-men. So sure are we that this is true that a syphilis census of the employees at the Massachusetts General Hospital—and we hope other hospitals also—and in the households of many of us who are particularly interested in the problem would show an amazingly high rate of incidence, because not only are people suffering from the disease not refused employment, but so far as possible employment is found for them.

How unfair is the refusal of a housewife to give work to a nursemaid with congenital syphilis, whose infectiousness was spontaneously cleared up perhaps twenty years ago while she was still an infant! Yet do we do all we can to explain such circumstances to the layman? A case in point, occurring about two years ago, remains in my memory, for though the girl was not badly marked, any third-year student would have recognized her Hutchinsonian teeth and hazy corneas. The prospective employer had a fit of terror and even threatened fumigation of the house after the first few minutes of the interview. The family physician seems to have agreed with the employer; her pediatrist tried to make her see the light, but to no avail. The young woman has since proved her lack of infectiousness by marrying and having a perfectly healthy baby.

Instruction must be given individually and not by printed directions or through a third person, for the problems to be met by the married man or woman and by the single man or woman, in the early case and in the late case, are quite different. Certainly the difference in the effect of the disease on the patient with congenital syphilis and one with the acquired form is as great as that in cases due to utterly different diseases. It is impossible to teach a patient all he should know about the problem during his first visit. One point that is important, however, is to make every effort to dissuade him from dusting off a copy of the so-called "doctor book" that was long treasured by his grandmother, giving lurid tales of the horrors of syphilis as they occurred seventy-five years ago when cases were recognized only by the ravages of the disease. Little can be offered a patient as to informative literature which is within his power to understand, for but little has been written for the layman. So the physician must be prepared to explain painstakingly various points of the disease, visit by visit, perhaps throughout the entire treatment. No matter how well informed the social worker of the clinic may be, it is necessary for the physician to go into detail on the first visit and have the social worker supplement his efforts, rather than merely give the patient the drug and leave the details to the social worker. The latter must be chosen with great care, and must be a person with whom both men and women will feel free to talk, one who is understanding and cheerful. This last attribute is absolutely essential, and realization of its value has been well expressed in the remark that one smile in the clinic is worth two social workers in the field.

464 Beacon Street.

Discussion

Dr. Ray L. Whitney, Boston: For my introduction to the discussion of this presentation of Dr. Cheever's, I shall quote Henry F. Stoll, who said, when he addressed the Section of Diseases of Children at the seventy-second annual session of the American Medical Association held in Boston in June, 1921: "For one not specializing in diseases of children to appear before a group of distinguished pediatricians merits an explanation, if not an apology. My presence here is due to the fact that within the last few years I have been consulted by a number of adults whose symptoms were due to prenatal syphilis unrecognized until irreparable damage had been done." In like manner my presence here today is due in part to the fact that through Dr. Cheever I have become interested in certain psychiatric aspects of the treatment of syphilis.

This paper of Dr. Cheever's is one which does not admit of argument, and its discussion from a psychiatric point of view has to be limited to the barest essentials. As a result of the more recent psychotherapeutic advances in the field of internal medicine, it has become apparent that the purely empirical procedures in the treatment of organic diseases may need to be supplemented in selected cases by psychotherapy, as for example in certain cases of peptic ulcer and mucous colitis. From the cases which Dr. Cheever cites one cannot but realize that psychic factors also play no insignificant part in the therapy of syphilitic diseases. As Dr. Cheever well says, there is more in the treatment of syphilis than "what can be pressed through a needle." Surgeons tell us that the preoperative patient who goes to the operating room fearful and apprehensive is more apt to have a stormy convalescence than the patient who passes through the same doors in an optimistic frame of mind. If, then, we are really to understand and treat the patient who has a so-called functional or an organic disease, we must see him as an individual who has problems of adjustment to the disease.

Syphilis, like pulmonary tuberculosis, cancer or the menopause, puts new problems before the individual. These problems are in the nature of emotional stresses, and may be of sufficient severity to be considered traumatic. They do not remain merely in the physical sphere, and need to be considered from the angle of their re-

percussions upon the total personality. Stokes, Kulchar and Pillsbury* aptly observed in their studies of urticaria some of the personality types and the manner in which they handled these traumas. They said: "There was a difference in the reactions of the man who exploded, philosophized the situation and calmed down, the man who sustained lasting regret, and the subjects who 'laughed it off'. . . . It is more the type of person than the type of stress and strain which appears in the background."

As Dr. Cheever points out, recently acquired syphilis and particularly congenital symptoms discovered in the juvenile period of life may be the source of an emotional shock which permeates the whole being and affects moral attitudes. From a psychotherapeutic standpoint this is a spiritual scar which ought to be preventable. This was recognized by Abner Post,† whom we all revered, who said as far back as 1914: "And now, this paper would be incomplete without a word as to the study of the disease [syphilis] as a whole. It is usually spoken of as a venereal disease and syphilis and gonorrhea are classed together. . . . They need to be studied as distinct diseases and not as evidences of immorality. The physician must do his share in their prevention and control quite as much as the moralist. . . .

"The desire to attach disgrace to all victims of the disease leads to concealment and difficulty in study."

As Dr. Cheever observes, much depends on the first consultation. Benefit or harm may come from it. It may seem to you that his paper is too sentimental, — I am sure he would not ask me to offer any apologies for it, — but it must be apparent to you that besides dealing with syphilis itself we are dealing with an individual who, in most instances, is highly charged emotionally because he has become infected. It must be remembered, too, that we need to treat the person behind his syphilis for these emotional changes, his attitudes toward his future, and so forth. Mistakes made at this time, for example provocative questions, can and do aggravate the patient's condition. It is essential to establish confidence in the patient that will enable him to trust us and tell us of his mental conflicts.

It is trite to say that the personality of the physician goes a long way toward effecting a healthy mental attitude in his patient.

*Stokes, J. H.; Kulchar, G. V., and Pillsbury, D. M.: Effect on the skin of emotional and nervous states: etiologic background of urticaria with special reference to the psychoneurogenous factor. Arch. Dermat. & Syph. 31:470-499, 1935.

†Post, A.: Mortality of hereditary syphilis. Boston M. & S. J. 170:113-116, 1914.

CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

ANTEMORTEM AND POSTMORTEM RECORDS AS USED IN WEEKLY CLINICOPATHOLOGICAL EXERCISES

FOUNDED BY RICHARD C. CABOT, M.D.

TRACY B. MALLORY, M.D., *Editor*

CASE 24441

PRESENTATION OF CASE

A fifty-nine-year-old married, native leather worker entered complaining of a mass in the epigastrium and abdominal swelling.

Four years before entry he noted a mass in the right upper quadrant. It slowly increased in size but was painless and not associated with jaundice or gastrointestinal symptoms. His physician advised x-ray treatment, but because of the absence of symptoms, he refused. Gradual swelling of the abdomen began one and a half years before entry. Six months later the ankles began to swell and caused pain on standing. The swelling gradually increased, and one month before entry the patient entered an outside hospital. An abdominal paracentesis there yielded clear, greenish fluid. Potassium iodide was given until the patient developed a severe rash.

During the last four years he had drunk 3 oz. of gin every night, but before that drank very little. He had had gonorrhea at the age of twenty-four. Fifteen years before admission he had had three eroded areas on his penis followed by a generalized skin rash; he did not have a sore mouth, enlarged lymph nodes or falling hair. He began to lose weight five years before entry, with a decrease of 33 pounds in a year, after which the process stopped.

Physical examination showed an elderly undernourished man with a large, rounded abdomen and edema of the ankles. The skin and sclerae showed slight but definite jaundice. Small telangiectatic areas were scattered over the entire body. Examination of the pupils and fundi was negative. The tongue was smooth and beefy. There was some flare to the lower rib margins, and the clavicular fossae were deep. Examination of the heart was negative. The blood pressure was 130 systolic, 70 diastolic. The diaphragms were high, especially on the right, but the lungs were negative. The abdomen was markedly distended, and there was a fluid wave, with dullness in the flanks. There was a mass barely palpable in the right upper quadrant, which moved with respiration. No lymph nodes could be palpated.

The temperature was 98°F., the pulse 70 and the respirations 20.

The urine contained bile and 10 to 20 white cells per high-power field. The blood showed a red-cell count of 3,880,000 with 70 per cent hemoglobin, and 8450 white cells with 88 per cent polymorphonuclears. Three stool examinations were guaiac positive, one negative. A van den Bergh was biphasic, and the blood serum contained 2.0 to 2.5 mg. per cent of bilirubin. The blood Hinton and Wassermann tests were positive. A bromsulfalein test of liver function showed 0 to 5 per cent retention after thirty minutes. An abdominal paracentesis produced 3500 cc. of thin, straw-colored fluid, which did not clot readily on standing. The specific gravity was 1.010; it contained 218 red cells and 336 white cells per cubic millimeter; no tumor cells were seen. The total protein content was 1.8 mg. per cent; a culture showed nonhemolytic streptococci. A Takata-Ara test was positive.

An x-ray film showed evidence of extensive esophageal varices and a small hernia of the stomach through the hiatus. A flat plate of the abdomen failed to show the spleen, but the liver appeared comparatively small. Chest films showed the diaphragm to be high in position. Linear markings at both lower lung fields indicated poor aeration. The heart was of normal size and shape. The aorta was markedly tortuous, slightly dilated and calcified.

On the third hospital day the liver edge and spleen could not be palpated. A vague mass was still present in the right upper quadrant. The vibratory sense was absent below the fourth dorsal vertebra. The right ankle jerk was greater than the left. During the next few days the abdominal fluid reaccumulated rapidly. On the ninth hospital day, a lumbar puncture showed normal dynamics. The total protein was 17 mg. per cent. A Wassermann test was weakly positive. The goldsol was 0122100000. On the ninth hospital day, five days after potassium iodide therapy had been begun, a rash simulating erythema multiforme appeared on the thighs. On the eleventh hospital day this eruption was purpuric in nature. That afternoon he suddenly vomited 400 cc. of fresh blood. The blood pressure dropped to 75 systolic, 45 diastolic. However, he felt better and the blood pressure soon rose. On the following day there was no further bleeding. His temperature varied from 98 to 101°F. during the first ten days, but after the eleventh day remained subnormal. His pulse ranged from 60 to 100, and the respirations were constantly at about 20. Although no further hemorrhages occurred, the patient became rapidly weaker and died on the fifteenth hospital day.

Differential Diagnosis

Dr. Chester M. Jones: There are certain things that are distinctly obvious and several other things that are not clear in this case. It seems perfectly evident that this man had a fatal liver condition as the basis of his illness. I think there is no reason to doubt that he had what I should classify as toxic cirrhosis and, in addition, very definite deficiency disease. The latter is the rule rather than the exception in advanced cirrhosis and usually represents the result of prolonged malnutrition. The deficiency disease was evidenced by the peripheral neuritis, loss of vibration sense, the edema, the red beefy tongue and the anemia. Vitamin B deficiency was undoubtedly a part of the picture in this particular instance, and the appearance of purpura suggests vitamin C deficiency, although the purpura may have been toxic in origin. At any rate it is obvious that serious malnutrition has been present for some time, and there may have been a substitution of alcohol for food, — a common finding in toxic cirrhosis, — with resulting changes in the peripheral nervous system, the liver and the blood. He had easily demonstrable esophageal varices, which could occur with only one condition I know, namely cirrhosis of the liver, usually classified as toxic or portal. This condition is most commonly associated with a high alcoholic intake, but a large proportion of patients do not use alcohol in any form. I have recently seen two or three cases where another type of cirrhosis initiated the patient's symptoms, and subsequently changes took place resulting in back pressure in the portal system with esophageal varices as a terminal result. Such an occurrence is not clearly mentioned in the literature, but I think we can find cases that show that it really takes place. On the law of chances, however, this patient has early toxic cirrhosis.

The findings of toxic cirrhosis, and I think this is in part a correct diagnosis, are of interest; but in addition we have the story of an epigastric mass which had been noticed by the patient four years previously. We do occasionally see patients with cirrhosis who are aware of a large liver and can feel the edge or a mass in the epigastrium. This may grow larger, or in many cases smaller, as the disease progresses. During four years this patient developed symptoms of hepatic insufficiency, and in addition ascites and peripheral edema. The vast majority of cases of liver disease will have ascites or edema, and a large number of them will have both findings. Usually the ascites precedes the edema, but there are a number of cases where the reverse is true, and the patients are treated primarily as those with cardiac disease.

We know this patient had had gonorrhea and primary lesions of syphilis, and we know that he had serological syphilis on entry. Furthermore, he was treated for syphilis for one month before entry, and it is of some interest that the mass which he noted four years before was not noticed when he came into the hospital. Failure to notice this mass may have been due to the fact that if it was the liver it had become smaller because of the normal course of events occurring in cirrhosis or because of specific iodide therapy. In the latter event, one must assume that the mass was gummatous. It is possible that the abdomen was so full of fluid that the mass could not be accurately palpated. The diaphragm was high by x-ray, and the liver and spleen were not made out. Although x-rays did not demonstrate either the liver or the spleen, the presence of ascitic fluid makes it very difficult to obtain good soft-tissue shadows, and therefore negative findings are not too important. One has to entertain the idea that he had syphilis and conjecture about a possible gumma, which was treated with reduction in the size of the liver and the development of portal obstruction — a rare occurrence. I do not believe that this is the logical explanation. I am inclined to think that he has two conditions in the liver: toxic cirrhosis, portal in type, and syphilis. We have recently had cases where these conditions coexisted. That is true, is it not?

Dr. Tracy B. Mallory: I so interpreted them, at any rate.

Dr. Jones: I believe that is a possibility here.

Why did he die? He could have died of hemorrhage. We know he vomited 400 cc. of blood, and quite possibly he oozed continuously from then on. Three out of four stool examinations were positive for occult blood. Possibly there was an interval when he was not bleeding. He could have died from malnutrition and hemorrhage alone. It is possible he may have had, as a terminal event, an atrophy of the liver; such a lesion could occur in the absence of jaundice or with slight jaundice. In a case of long-standing cirrhosis, one should mention that cancer can be superimposed — a primary hepatoma. But in these cases one usually has an easily palpable liver or mass demonstrable by x-ray, and the diaphragm instead of being smooth shows projections due to pressure from an irregular liver surface. However, I do not believe he has cancer of the liver. The skin rash that he developed might well have been an iodide rash; or it might have been a toxic rash in a patient who had serious intrahepatic disease. The liver should show portal or toxic cirrhosis with obstruction in the portal system and esophageal varices. He certainly had a deficiency syndrome. He may well have shown a syphilitic process in

the liver which had been more or less successfully treated and there was possibly, but not probably, some atrophy in addition.

DR. AUBREY O. HAMPTON: I do not see any evidence of metastatic malignancy in the lung. The lines that were described in both lower lobes are undoubtedly due to atelectasis.

DR. JONES: Due to high diaphragm with ascites?

DR. HAMPTON: Yes. He did have dilated loops of small bowel, which were not mentioned in the record, that we interpreted as due to ileus. I should like to say again that we cannot differentiate organic obstruction and ileus with the routine film of the abdomen. We can say that the small bowel is filled with gas and dilated and that these findings may be due to organic obstruction, peritonitis or ileus. In this case there was sufficient reason for us to say there was ileus, because of ascites and varices of the esophagus. The varices are very obvious in practically all the films taken. They extend up to about the middle of the esophagus and are visible even in the upright position.

I think Dr. Jones is correct in regard to the comment on whether the liver and spleen were enlarged. We cannot see these shadows in the presence of ascites. The gas-filled bowel is relatively high on the right, and that is why we made the statement that the liver was not enlarged and might be small.

DR. FULLER ALBRIGHT: There is one point about the physical examination that should be cleared up. He did have a mass in the epigastrium on admission. A slight irregularity could be felt. Everyone agreed on that.

CLINICAL DIAGNOSES

Cirrhosis of liver with secondary carcinoma of liver.
Syphilis of liver (hepar lobatum).
Perforation of esophageal varices.

DR. JONES' DIAGNOSES

Toxic (portal) cirrhosis.
Syphilis of liver.

ANATOMICAL DIAGNOSES

Syphilis of liver, gummatous.
Esophageal varices.
Splenomegaly.
Ascites.
Peripheral edema.
Arteriosclerosis, generalized, mild.

PATHOLOGICAL DISCUSSION

DR. MALLORY: I was rash enough in this case to deliver a clinical lecture to the medical students before we started the autopsy. I was told that the primary cause of death was thought to be syphilis of the liver, and I said that it practically never produced portal obstruction or the severe symptoms of hepatic insufficiency which this patient had and that he must have something else to account for the picture. The autopsy showed a very large gumma almost completely replacing the left lobe of the liver and a very granular right lobe. I still thought I was right. I admitted the gumma but pointed out that he obviously had a cirrhosis as well. The sections have now come through and show multiple miliary gummas throughout the liver with focal areas of scarring but nothing remotely to suggest alcoholic or portal cirrhosis. I am quite sure after studying them that he suffered from nothing but syphilis of the liver, still actively progressive at the time of his death.

DR. ALBRIGHT: We looked up the question of varices accompanying syphilis of the liver and found you can get them.

DR. MALLORY: We have had twenty cases of hepar lobatum and have found varices in only two.

DR. HAMPTON: What was the size of the left lobe — larger than normal?

DR. MALLORY: No, smaller than normal.

DR. JONES: I think you can have varices from any cause that cuts down on the portal circulation.

DR. MALLORY: The extent to which the scars of gummas can deform a liver without producing a portal obstruction is quite remarkable. This is the exception to the rule.

DR. GEORGE W. HOLMES: How large was the spleen?

DR. MALLORY: It weighed 400 gm. It was enlarged to just about the limits of palpability. The immediate cause of death was rupture of the varices.

CASE 24442

PRESENTATION OF CASE

First admission. A sixty-four-year-old married Irish stone mason was first admitted complaining of severe gripping pain in the right abdomen and chest of two days' duration.

Four years prior to admission he was found to have serological syphilis, and bismuth therapy had been given for more than three years at an outside dispensary. One year before entry he entered the Out Patient Department complaining that hiccoughing had developed five weeks previously, which lasted for two weeks, and was followed by a brief period of coughing up thick brown phlegm. This was followed by two weeks

of cramplike pain in the left flank. During the entire illness he had lost 20 pounds in weight. X-ray films showed a large aneurysm involving the descending aorta just below the arch. He was discharged for further treatment at the outside dispensary. Forty-eight hours before admission, immediately after an evening meal, there was a sudden onset of severe, gripping right lower-quadrant pain beginning just to the right of the navel and rapidly spreading upward to the right anterior chest. It was so severe that it caused him to collapse to the floor. There was radiation of pain up and down between the right chest and right lower quadrant. A hypodermic injection given by his physician relieved him. On the following day he had fleeting girdle pains and another severe episode of shocking pain throughout the right side. He was brought in by ambulance and found to be in a state of shock. The blood pressure was low, and the neck veins distended. He was dyspneic but the heart and lungs were not remarkable. Because of poorly localized pain in the right upper quadrant, a tentative diagnosis of cholecystitis was made on admission.

Twenty years prior to entry an apparent primary syphilitic lesion on the penis was treated by cautery.

Physical examination showed a well-developed, slightly undernourished, dyspneic man sitting up in bed. There was a frequent harsh cough productive of blood-streaked sputum. The pupils were round and equal and reacted to light and accommodation. The neck veins were markedly distended. Dullness and a few rales were elicited at the right base posteriorly. The supracardiac dullness was increased, and the heart was slightly enlarged downward and to the left. There was a faint apical systolic murmur and a moderately loud, blowing diastolic murmur at the base, with a diastolic thrill. There were frequent extrasystoles. The blood pressure was 120 systolic, 60 diastolic, in each arm. There was tenderness in the abdomen, without spasm. No masses or organs were palpated. The extremities were negative.

The temperature was 98.8°F., the pulse 110 and the respirations 30.

Examination of the urine was negative. The blood showed a red-cell count of 3,500,000 with 60 per cent hemoglobin. The white-cell count was 11,700 with 77 per cent polymorphonuclears. The nonprotein nitrogen of the serum was 53 mg. per cent. A van den Bergh was normal, indirect. The serum phosphatase was 7.76 units per cent. A blood Hinton test was positive, a Wassermann test negative. A stool examination was negative. An electrocardiogram was negative except for a low T_1.

X-ray films showed that the aneurysm of the descending aorta had markedly enlarged. An aneurysm was also seen involving the beginning of the arch, as well as the innominate artery. A soft-tissue mass, which appeared to be a third aneurysm involving the abdominal aorta, was visible below the diaphragm on the left side. Both pleural cavities contained a small amount of fluid, and there was poor aeration of the right lower lobe. There was evidence of Paget's disease of the fourth lumbar vertebra, right pelvis and femur.

On the second hospital day the patient had vague pains in the chest and abdomen, as well as a harassing cough and difficulty in swallowing food. He improved only slightly. On the twelfth hospital day he was discharged to his physician.

Final admission (one week later). After discharge he developed persistent hiccoughs and vomited all food. His dyspnea increased, and he became dehydrated and lost more weight.

Physical examination on admission showed a large area of tympany at the left base, where breath sounds were diminished but fremitus unaffected. The heart seemed definitely displaced to the right. The physical examination was otherwise the same as at discharge. The temperature was 101°F., the pulse 100, and the respirations 24.

On the afternoon of the day of admission he began to vomit gross blood, raising 800 cc. within half an hour. The bleeding stopped, and the following morning his blood pressure was 118 systolic, 60 diastolic. Then fresh hemorrhages began, and he raised about a quart of blood within a few minutes. There was no obvious respiratory difficulty, but the blood was slightly frothy. He rapidly failed and died within fifteen minutes.

Differential Diagnosis

Dr. Howard B. Sprague: In this case they apparently give us the diagnosis but withhold the mechanism of his death, and this leads us to a good deal of speculation. The man was known to have syphilis, having been treated in the method of many years ago by local extirpation of the primary lesion. It was not until he was sixty years old that this was discovered, and during these twenty years he had developed a great many abnormalities in his cardiovascular system. If we start with his heart we find that he had a somewhat enlarged one with a loud diastolic murmur at the base and a diastolic thrill. That in a syphilitic patient should spell aortic regurgitation. The degree of regurgitation is perhaps not very marked,

as judged by the size of his heart and the diastolic pressure of 60. In other words, the changes were more marked beyond the aortic orifice and the ostia of the coronary arteries, with the development of multiple aneurysms of the aorta. He had one aneurysm that roentgenologically is seen to involve the beginning of the arch and the innominate artery, but in this case we have no evidence that the aneurysms affected the blood pressure on one side or the other, that is, his blood pressure was equal on both sides. Beyond that we come to another aneurysm which was discovered in the descending aorta, just below the arch, and still farther along the course of the aorta we come to some sort of soft-tissue mass, possibly below the diaphragm on the left side, which appears to be another aneurysm. The second x-ray shows that the aneurysm in the descending aorta had markedly increased in size between the two examinations. Now if we have three aneurysms we may have more that may not be demonstrated. All his symptoms and signs are consistent with pressure from these various dilatations. The fact that he came in the first time after having had a severe attack of pain with low blood pressure and distended neck veins, makes me think that the cause was a pressure phenomenon with distention of veins in the neck rather than simply a congestive failure.

What happened in his abdomen in the early part of the story? The attack came two days before he entered the hospital and was characterized by severe pain beginning to the right of the navel and apparently spreading down to the right lower quadrant and up to the right chest. It was very severe, associated with some degree of shock, and was repeated again on the following day. Also, he had hiccoughing, which would indicate diaphragmatic irritation.

I shall ask Dr. Hampton to look at the x-rays and perhaps point out the exact location of the aneurysm in order to help us decide about the pressure.

X-Ray Interpretation

Dr. Aubrey O. Hampton: There is an interval of a year between the first and second examinations. The first examination showed a very obvious aneurysm of the descending aorta, extensive calcification of the entire ascending and transverse aorta and calcification in the walls of the aneurysm extending down to the diaphragm. This calcium makes it possible to measure the aneurysm very accurately. I should say it was 10 cm. at its widest point. His ascending aorta does not show the bulge to the right which you would expect if it were dilated. He had a definite difference in the radiance of the lower lobes at the first examination. He also had mottled density in the left upper lobe and right apex. His heart was not grossly enlarged. You could assume from this examination that he did not have aortic regurgitation of long standing and that he did not have an aneurysm of the ascending portion of the aorta at the first examination. His chief difficulty was compression of the left lower bronchus from aneurysm of the descending aorta. This is obstructive emphysema here in the left lower lobe. The lung is brighter and more completely aerated than the corresponding lobe on the opposite side. He could take air into the lung but could not get it out. This finding is much more marked after a period of a year. No shadows of blood-vessels are seen, and there appears to be just one sac of air.

Dr. Sprague: Can you say that the air is in the lung rather than in the pleural cavity?

Dr. Hampton: You should be able to, although I admit there might be some confusion about it. This smooth round margin at the costophrenic angle would not be present if there were air in the pleural cavity.

There was another change during the one-year period. Aneurysms of the ascending and the arch portions of the aorta developed. As I remember this case it seemed to me to be a very rapid change.

There is one further point in this last film. This is a gas-filled stomach. This is the air-containing sac of the left lower lobe and this shadow pushing in on the stomach is continuous with the aneurysm of the thoracic aorta. So he also had evidence of aneurysm of the abdominal aorta. He had multiple aneurysms of the descending portion, one of which appears to be below the diaphragm.

Differential Diagnosis (continued)

Dr. Sprague: We have had some important information from the x-ray studies. It would seem that the attack he had a year before his last entry, the abdominal pain, the hiccoughing, and so on, could be explained as part of the process of the rapid development and dilatation of these aneurysms, including in this particular case the abdominal aneurysm, the extent of which we do not know. We cannot see the upper end of it here. And because of the fact that he recovered at that time we can assume that no rupture occurred and that he did not have any seepage from a localized collection of blood. In the final episode we have cough and blood-streaked sputum, which could be well explained by the pressure we have seen on the left primary bronchus. We have distended neck veins, which again could be a pressure phenomenon from the aneurysm of the arch and the first portion of the aorta.

Then we have the final episode which is now explained as being due not to what we might have thought earlier, that is, air's getting into the pleural cavity with hemopneumothorax but to pressure and obstructive emphysema. At the end we also have the vomiting of gross blood.

The ordinary final episode of a rapidly increasing aneurysm is rupture. In this case a large amount of fresh blood found its way into the gastrointestinal tract. It is conceivable that he had pressure of a type to cause venous congestion there and rupture of a varix of the stomach or the esophagus, or it is possible that an aneurysm actually ruptured into the lower end of the esophagus or stomach. The fact that the hemorrhage was not immediately followed by death is somewhat against rupture of the aneurysm into the esophagus and in favor of venous bleeding such as you get in cirrhosis of the liver. The bleeding stopped and then recurred. But this man had so much in the way of pressure disturbance in his respiratory mechanism and also involvement of the heart and coronary ostia that the added difficulty from the bleeding was apparently enough to kill him. It is a good deal commoner to observe rupture of aneurysms into the bronchial tree than into the gastrointestinal tract, and the statement that the blood was slightly frothy might suggest such an occurrence. However, the history states that the blood was "vomited" rather than coughed up, and I believe that the hemorrhage into the esophagus or stomach was due to a ruptured aneurysm or a ruptured varix.

Clinical Diagnoses

Cardiovascular syphilis with multiple aneurysms.
Syphilitic heart disease and aortic regurgitation.
Rupture of aneurysm into lungs or gastrointestinal tract.

Dr. Sprague's Diagnoses

Syphilitic cardiovascular disease.
Syphilitic aortitis with multiple aneurysms and aortic regurgitation.
Hemorrhage into the stomach or esophagus from ruptured aneurysm or varix.

Anatomical Diagnoses

Syphilitic aortitis with multiple aneurysms.
Rupture of aneurysm into mediastinum and esophagus.
Hematoma of stomach.
Pulmonary tuberculosis, healed, apical.
Bronchopneumonia, right base.
Paget's disease.
Arteriosclerosis, generalized.

Pathological Discussion

Dr. Benjamin Castleman: This man had a small heart, and the aortic valve was perfectly normal, showing no evidence of separation at the commissures and certainly nothing to suggest to us that there was any degree of regurgitation. The aorta showed the aneurysmal dilatations that have been described. In the region of the junction of the middle and lower thirds of the esophagus there was a rupture of the aneurysm into the esophagus. It must have been a very slow perforation, really a seepage, because the blood had worked down through the wall of the esophagus and through the mediastinum into the abdomen and had produced a large hematoma, measuring about 7 cm. in diameter and filled with clotted blood, which pressed against the stomach in the region of the cardia.

Dr. Sprague: A dissecting aneurysm of the esophagus?

Dr. Castleman: Yes. The hematoma was in the wall of the stomach and simulated an aortic dissecting aneurysm. You could see serosa on one side and mucosa on the other — quite an unusual picture. Histologically, the aorta showed definite evidence of syphilis.

The other interesting feature of this case concerned the lungs. The trachea and major bronchi were markedly flattened posteriorly, especially in the region of the left lower-lobe bronchus, by the large aneurysm. Perhaps Dr. Hampton would like to show the postmortem films at this point.

Dr. Hampton: This postmortem film shows much more obviously the emphysematous changes at the left base than the last films taken in life. The diaphragm is markedly depressed, and the lung tissue shows no blood-vessel shadows. The left lower lobe appears to consist of a single sac of air. There is also some mottled density throughout the other lobes, characteristic of bronchopneumonia.

It is interesting to compare the antemortem and postmortem films. It brings up a subject we have discussed before. Is the measurement of the aorta at autopsy of much value? It is of course normally an elastic organ and therefore shrinks when the blood pressure drops to zero. With aneurysmal dilatation you would not expect much elasticity, and yet the aneurysms appear much smaller in the postmortem than in the antemortem films.

Dr. Sprague: That seems particularly surprising since their walls were calcified.

Dr. Castleman: Most of the left lower lobe was composed of a large thin-walled cyst measuring 19 by 17 by 5 cm. and corresponding to the increased radiance of this lobe on the x-ray film.

As Dr. Hampton pointed out the increasing pressure upon the bronchus by the aneurysm caused progressive obstruction to expiration and a true obstructive emphysema. Could the aneurysm alone have caused the development of the cyst? I suppose it is theoretically possible. However, the presence of a fairly diffuse, moderately severe emphysematous process throughout both lungs makes it quite likely that some degree of emphysema antedated the aneurysm. The latter undoubtedly aggravated the condition in the left lower lobe, to produce the large bleb. In addition there was old tuberculosis in the upper lobes, and bronchopneumonia in the right upper lobe.

DR. SPRAGUE: Was there any sign of perforation into the pulmonary tree?

DR. CASTLEMAN: No; we looked carefully for that because of the statement regarding frothy fluid.

DR. SPRAGUE: There is also no explanation of the previous attack of abdominal pain?

DR. CASTLEMAN: No.

DR. SPRAGUE: The question was brought up whether the involvement of the vertebrae with Paget's disease could have been responsible for the pain.

DR. CASTLEMAN: I do not believe so. You usually do not get pain from Paget's disease in the vertebrae.

DR. SPRAGUE: If it is true that there was a diastolic murmur with a thrill in the aortic area, it must have meant something.

DR. CASTLEMAN: It could have been due to aneurysm.

DR. SPRAGUE: It might have been a functional aortic regurgitation during life. There is so much change in aortic size between life and death that dilatation might have been present during life, and if it were, there might have been considerable functional regurgitation.

DR. CASTLEMAN: As you say, his blood pressure certainly was not that of the usual case of aortic regurgitation.

DR. SPRAGUE: No. Furthermore, this murmur was not described on his first admission: "The heart was not remarkable." It was heard only after the development of an aneurysm of the ascending aorta, with dilatation of the innominate. All we can say is that it was an extremely rapid progress for an aneurysm.

DR. CASTLEMAN: A somewhat similar case of dissection of blood from a ruptured aortic aneurysm through the esophageal wall into the stomach has just been reported by Moser* in the Transactions of the Pathological Society of Philadelphia.

*Moser, H.: Aortic aneurysm with rupture into the stomach. Arch. Path. 26:907, 1938.

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The Journal does not hold itself responsible for statements made by any contributor.

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POSTGRADUATE ASSEMBLY

During the past week every physician in New England has received an official program of the New England Postgraduate Assembly, which will be held in Sanders Theater, Harvard University, Cambridge, on November 15 and 16. The committee respectfully requests that the application blank, which is on the flap of the envelope, be promptly filled out and returned. It is self-addressed and requires no postage. The complete program of the assembly also appears on page 723 of this issue of the *Journal*. As soon as your application has been received there will be mailed to you a badge, dinner ticket, windshield sticker and whatever luncheon tickets you may have ordered and paid for.

The purpose in sending these prior to the date of the meeting is for your convenience. The badge that is being issued is your ticket of admission to Sanders Theater. Keep it and wear it on November 15 and 16. It will save you the trouble of registering at the time of the assembly and possibly the annoyance of having your original application checked. The dinner ticket admits you to Memorial Hall on Tuesday evening. The windshield sticker will entitle you to reasonable consideration from the Cambridge Police Department. Failure to use it on your car may cause you the annoyance of being tagged for overtime parking. If you wish to have luncheon on one or both days at Memorial Hall, be sure to fill in the appropriate blank or blanks on the application. An estimate of the attendance must be given to the caterer on the preceding day, and, when this number has been reached, the sale of tickets will stop.

In order that you may get the fullest enjoyment from the assembly, you are urged to send in your application for registration at the earliest convenient moment.

THE APPOINTMENT OF DR. PERKINS

His Excellency, Governor Hurley, and the Governor's Council have acted wisely in appointing Dr. Clifton Todd Perkins as commissioner of the Department of Mental Health. Dr. Perkins is well qualified for the position. He received his A.B. from Bates College in 1922 and his M.D. from Boston University School of Medicine in 1926 and then completed an internship and residentship at the Massachusetts Memorial Hospitals, during which time he took an active part in clinical investigations on contagious diseases. From 1928 to 1930 he was senior physician at the Worcester State Hospital, rising to the positions of acting assistant superintendent and assistant superintendent; he held the latter position from 1932 to 1935. While there he carried out and published a report on special studies in general paresis.

On December 1, 1935, Dr. Winfred Overholser called Dr. Perkins to the State House as assistant to the commissioner of mental diseases. On June 23, 1937, he was appointed assistant commissioner, and on May 6, 1938, acting commissioner. From 1932 to 1936 he was an instructor in psychiatry at

Boston University School of Medicine and from December, 1935, to July, 1937, he was rated as a state expert for examination of insane criminals.

He is a member of the Massachusetts Medical Society, the American Medical Association, the Massachusetts Psychiatric Society, the New England Society of Psychiatry and the American Association on Mental Deficiency and a fellow of the American Psychiatric Association.

Because of Dr. David L. Williams's retirement on account of ill health on May 20, 1937, and Dr. Joseph E. Barrett's resignation on June 1, 1937, to become the commissioner of mental diseases in Michigan, Dr. Perkins was left in complete charge of the Department of Mental Diseases. Consequently, through a very trying period, Dr. Perkins was forced to shoulder the responsibilities of the department without adequate help, owing to conditions which prevented the appointment of the usual assistants. By working long hours and without vacations, he sometimes carried the work of five men at a time when as many as four investigating committees were inquiring about the department and certain of the hospitals. In spite of all this, he has prepared a long-term building program, made a special study of medical care and of food and has accomplished much in the way of general improvements. His diligence, foresight and good judgment have gained the admiration and esteem of each of the former associate commissioners as well as of the Governor, and his appointment as commissioner to the lately reorganized Department of Mental Health is a just reward. With adequate assistants, Dr. Perkins will soon be able to regain for this department the prestige it formerly enjoyed throughout the United States.

A MEDICAL PLATFORM

THE following is an excerpt from a letter written by a state senator to the physicians of his constituency:

> In 1936 I pledged myself to the following "Medical Platform":
>
> 1. The continued betterment of the standard of medical practice.
> 2. The control of cultism by a single standard of examination for license to practice.
> 3. Opposition to all forms of compulsory health-insurance schemes until it can be shown that they are to the best interests of the profession and public.
> 4. The preservation of the sacred right of an individual to go to a physician of his own choice.
> 5. The furthering of legislation which will adequately stop loopholes in the existing insurance laws which deprive the physician of his just compensation.
>
> I have faithfully kept this pledge, and I shall continue to keep it until you and your profession direct otherwise.

If more of the members of the Legislature were to adopt such a platform, the efforts of organized medicine to improve medical standards and medical care in Massachusetts would undoubtedly be more successful than they have been in the past. Any candidate who so expresses his ideas in regard to medical practice deserves the unqualified support not only of the medical profession but also of the public.

MASSACHUSETTS MEDICAL SOCIETY

NEW ENGLAND POSTGRADUATE ASSEMBLY

SANDERS THEATER, HARVARD UNIVERSITY, CAMBRIDGE
NOVEMBER 15 AND 16

Every doctor in New England is cordially invited to attend the New England Postgraduate Assembly, sponsored by the Massachusetts Medical Society, to be held in Boston on November 15 and 16, 1938. The speakers are all prominent educators as well as successful practitioners in their chosen fields of medicine. This is an unusual opportunity for New England physicians to obtain the latest and best information in the diagnosis and treatment of many diseases. The list of guest speakers is as follows:

DR. FRANCIS G. BLAKE, New Haven, Connecticut.
Sterling Professor of Medicine, Yale University School of Medicine, and physician-in-chief, New Haven Hospital.

DR. LOUIS A. BUIE, Rochester, Minnesota.
Professor of proctology, Mayo Foundation for Medical Education and Research, Graduate School, University of Minnesota Medical School, and chief, Section on Proctology, Mayo Clinic.

Dr. William L. Estes, Jr., Bethlehem, Pennsylvania.
Chief surgeon, St. Luke's Hospital.

Dr. Robert T. Frank, New York City.
Clinical professor of gynecology, Columbia University College of Physicians and Surgeons, and consulting gynecologist, Mount Sinai Hospital.

Dr. Alvah H. Gordon, Montreal.
Professor of medicine, McGill University, and physician, Montreal General Hospital.

Dr. Perrin H. Long, Baltimore.
Associate professor of medicine, Johns Hopkins University School of Medicine, and assistant and associate physician, Johns Hopkins Hospital.

Dr. Louis H. Nahum, New Haven, Connecticut.
Assistant professor of physiology, Yale University School of Medicine.

Dr. Hubley R. Owen, Philadelphia.
Professor of clinical surgery, Woman's Medical College of Pennsylvania, and surgeon, Philadelphia General Hospital and Hospital of the Woman's Medical College of Pennsylvania.

Dr. Harvey B. Stone, Baltimore.
Associate professor of surgery, Johns Hopkins University School of Medicine, and visiting surgeon, Johns Hopkins Hospital.

Dr. Benjamin P. Watson, New York City.
Professor of obstetrics and gynecology, Columbia University College of Physicians and Surgeons, and director, Sloane Hospital for Women.

At the dinner on Tuesday evening, November 15, Dr. Warren F. Draper, assistant surgeon general of the United States Public Health Service, will speak on "Current Phases of the Problem of Medical Care." Rapid changes in the field of governmental participation in medical care make this topic one of absorbing interest, and to hear directly from a full-time governmental physician who understands the attitude of organized medicine should prove to be extremely valuable. Dr. Roger I. Lee will speak on "The Changing Private Practice of Medicine." The dinner will be held in Memorial Hall, Harvard University, at 7:00 p. m.

The registration fee for the Assembly is three dollars; this includes admission to all sessions and to the dinner. On Tuesday, November 15, and Wednesday, November 16, luncheons will be served in Memorial Hall, Harvard University, at seventy-five cents each. Memorial Hall is in the same building as is Sanders Theater where the assembly will be held. Reservations for luncheons must be made in advance.

PROGRAM

Tuesday, November 15

morning session

Presiding Chairman: Dr. Frank R. Ober.

10:00 Dr. Hubley R. Owen. "What Are the Duties and Responsibilities of the General Practitioner in the Treatment of Fractures? Unrecognized Fractures."

10:30 Dr. Alvah H. Gordon. "The Physician's Interest in Gallbladder Disease."

Intermission

11:15 Dr. Harvey B. Stone. "Preoperative and Postoperative Preparation of Surgical Patients."

11:45 Dr. Perrin H. Long. "The Practical Interpretation of Laboratory Bacteriological and Immunological Diagnoses in Relation to Infectious Diseases."

12:15 Luncheon: Memorial Hall.

afternoon session

Presiding Chairman: Dr. George R. Minot.

1:45 Dr. Hubley R. Owen. "Transportation of Injured and Emergency Treatment of Fractures With Special Reference to Fractures of the Long Bones and Fractures of the Spine."

2:15 Dr. Francis G. Blake. "The Value of Antitoxin in Scarlet Fever."

2:45 Dr. Louis A. Buie. "Office Methods of Diagnosis of Anal Diseases."

Intermission

3:30 Dr. Louis H. Nahum. "The Etiology and Treatment of the Cardiac Arrhythmias."

4:00 Dr. Robert T. Frank. "Endocrinology as Now Practiced."

4:30 Dr. William L. Estes, Jr. "Acute Traumatic Lesions of the Abdomen."

7:00 Dinner. Speakers, Dr. Warren F. Draper and Dr. Roger I. Lee.

Wednesday, November 16

morning session

Presiding Chairman: Dr. Elliott P. Joslin.

9:00 Dr. Francis G. Blake. "The Importance of Time in Serum Therapy."

9:30 Dr. William L. Estes, Jr. "The Use of Antiseptics in the Treatment of Open Wounds."

10:00 Dr. Alvah H. Gordon. "Mental Complications in Heart Disease."

10:30 Dr. Robert T. Frank. "The Female Sex Hormones."

Intermission

11:15 Dr. Benjamin P. Watson. "When Is Cesarean Section Necessary and When Is It Unjustified?"

11:45 Dr. Perrin H. Long. "The Clinical Use of Sulfanilamide in the Treatment of Bacterial Infections."

12:15 Dr. Harvey B. Stone. "Symptoms and Signs of Intestinal Obstruction."

12:45 Luncheon: Memorial Hall.

afternoon session

Presiding Chairman: Dr. Arthur W. Allen.

1:45 Dr. Louis A. Buie. "Treatment of Common Anal Diseases."

2:15 Dr. Alvah H. Gordon. "The Diagnosis of Diseases With Coincident Enlargement of the Liver and the Spleen."

2:45 Dr. Benjamin P. Watson. "The Treatment of Delayed Labor."

Intermission

3:30 Dr. Perrin H. Long. "The Uses of Newer Derivatives of Sulfanilamide in the Treatment of Bacterial Infections."

4:00 Dr. Hubley R. Owen. "Skull Fractures and Concussion."
4:30 Dr. Louis A. Buie. "The Origin and Treatment of Anal Fistulas." (Moving Picture Film.)

It will greatly aid the committee if everyone who plans to attend will fill out the application blank contained in the program recently mailed to all physicians in New England and return it to the committee at once. Each space should be filled, including the name and address; all this information is of extreme importance to the committee.

FRANK R. OBER, *Chairman,*
LEROY E. PARKINS, *Secretary.*
Committee on Postgraduate Instruction,
Massachusetts Medical Society.

SECTION OF OBSTETRICS AND GYNECOLOGY

RAYMOND S. TITUS, M.D., *Secretary*
330 Dartmouth Street
Boston

SPONTANEOUS RUPTURE OF THE UTERUS IN SCAR OF PREVIOUS CESAREAN SECTION — TWIN PREGNANCY

Mrs. L. R., a thirty-eight-year-old gravida IV, thirty-three weeks pregnant, complained of pain on May 16, 1934, and was seen immediately.

Her family and past histories were essentially negative. Her three previous pregnancies had been terminated by cesarean sections, the indication for the first cesarean having been an acute toxemia. Catamenia began at thirteen, were regular with a twenty-eight-day cycle, and lasted four days. Her last period began September 8, 1933, making her due for delivery June 15.

The present pregnancy was uneventful until April 13 when an x-ray showed a twin pregnancy. Because of the size of the uterus at this time and because of her previous cesarean sections, she was advised to enter the hospital and remain there until delivered. This she did on April 29. After admission she was matched and grouped for possible transfusion. Her stay in the hospital was uneventful until the date above mentioned. Examination at this time showed the uterus to be spastic, and because of the pain and because of the tenseness of the uterus, it was felt that the placenta was separating or that the scar in the uterus from the previous cesarean sections was rupturing. Immediate operation was deemed necessary.

A series of selected case histories by members of the section will be published weekly.
Comments and questions by subscribers are solicited and will be discussed by members of the section.

Under nitrous-oxide, oxygen and ether anesthesia the abdomen was opened through the previous scar. As the peritoneum was exposed there was a darkened area underneath, and as it was opened, a large amount of bloody fluid escaped. It was found that the previous cesarean scar had ruptured throughout its entire length. A female child in good condition, weighing 6 lb. 4 oz., was easily delivered, and the placenta followed immediately. The other child, a male, weighing 6 lb. 2 oz., which with its placenta was found to be lying free in the peritoneal cavity, was dead. The uterus was repaired in routine fashion, and sterilization by the Bishop technic was done. There was a large amount of blood and amniotic fluid and several large clots in the abdominal cavity. These were sponged out, and the abdomen closed in layers. As the patient's pulse was 140, and the blood pressure 114 systolic, 70 diastolic, no postoperative transfusion was given.

For the first few days after the operation there was a moderate amount of distention and more than a moderate amount of vomiting, which was relieved by stomach lavage. After the fifth day her convalescence was uninterruptedly normal.

Comment. In this case, the twins plus the abnormal amount of amniotic fluid simply distended the uterus beyond its normal ability to stretch, and rupture in the scar followed. It was fortunate that the patient was in the hospital at the time; the one living baby undoubtedly resulted from immediate operation, and if the rupture had occurred at the patient's home, the mother's life might have been lost. Hysterectomy was not performed because it was believed that the rent in the uterus could be closed successfully by resuturing and that the condition of the patient did not warrant the added risk of taking out the uterus. The operation of sterilization was certainly justified because the patient had had three cesarean sections and was thirty-eight years old.

CLINICAL POSTGRADUATE INSTRUCTION

The medical profession of Massachusetts will be interested to know that clinical postgraduate teaching is now available in gonorrhea and syphilis. The Massachusetts Medical Society, the Massachusetts Department of Public Health and the United States Public Health Service have taken this progressive step to meet the demands for practical service to practitioners. These clinics will be given in Boston and Springfield so that no one will have far to travel.

Any physician who desires to attend one or more

times is welcome at any of these clinics. Individual needs may be discussed with the instructor in charge, and one may register for a day or for a series of clinics. Physicians should feel free to call or visit any of these hospitals and ask for the teaching clinic for gonorrhea or syphilis. This instruction is free, since all expenses are met by the above organizations. Information may be obtained from the clinic by letter or telephone.

The schedule of clinics is as follows:

GONORRHEA POSTGRADUATE TEACHING CLINICS

From November 1, 1938, and continuing for twenty-five weeks

PLACE	TIME
Boston Dispensary (limited to five physicians per day).	Wednesday and Thursday, 9:30 a. m.-12 noon.
Springfield Hospital (limited to three physicians per day).	Tuesday, 4:30 p. m.-6:30 p. m. (men). Thursday, 4:30 p. m.-6:30 p. m. (women and children).

SYPHILIS POSTGRADUATE TEACHING CLINICS

From November 1, 1938, and continuing for twenty-five weeks

PLACE	TIME
Out Patient Department, Massachusetts General Hospital (limited to five physicians per day).	Wednesday and Thursday, 10 a. m.-12 noon.
Springfield Hospital (limited to three physicians per day).	Tuesday, 4:30 p. m.-6:30 p. m. (men). Thursday, 4:30 p. m.-6:30 p. m. (women and children).

INSTRUCTORS IN CHARGE

Gonorrhea. Boston Dispensary, Dr. Oscar F. Cox, Jr.; Springfield Hospital, Dr. Edward C. Sullivan.

Syphilis. Massachusetts General Hospital, Dr. C. Guy Lane; Springfield Hospital, Dr. Edward C. Sullivan.

MEDICAL POSTGRADUATE EXTENSION COURSES

The following sessions, given by the Massachusetts Medical Society in co-operation with the Massachusetts Department of Public Health, the United States Public Health Service and the Federal Children's Bureau, have been arranged for the week beginning November 7:

BRISTOL SOUTH (New Bedford Section)

The course will be omitted November 11 because of Armistice Day.

ESSEX NORTH

The course will be omitted November 11 because of Armistice Day.

ESSEX SOUTH

Tuesday, November 8, at 4:00 p. m., in the Nurses' Home of the Salem Hospital, Salem. Subject: Gonorrhea — Modern Treatment of Gonorrhea. Instructor: George C. Prather. Walter G. Phippen, *Chairman*.

HAMPSHIRE

Wednesday, November 9, at 4:15 p. m., in the Nurses' Home of the Cooley Dickinson Hospital, Northampton. Subject: Anemia — Modern Methods in Diagnosis and Treatment of Blood Dyscrasias. Instructor: Clark W. Heath. Warren P. Cordes, *Chairman*.

NORFOLK SOUTH

Monday, November 7, at 8:30 p. m., at the Quincy City Hospital, Whitwell Street, Quincy. Subject: The Toxemias of Pregnancy. Instructor: Foster S. Kellogg. David L. Belding, *Chairman*.

PLYMOUTH

Tuesday, November 8, at 4:00 p. m., in the Rosa Field Nurses' Home, Brockton Hospital (rear of hospital), Brockton. Subject: Heart Disease — The Treatment of "Heart Attacks" or "Cardiovascular Emergencies." Instructor: Sylvester McGinn. Walter H. Pulsifer, *Chairman*.

WORCESTER

Tuesday, November 8, at 8:30 p. m., in the Nurses' Home of the Milford Hospital, Milford. Subject: Surgical Complications in Pregnancy. Instructor: Raymond S. Titus. Joseph Ashkins, *Chairman*.

WORCESTER NORTH

The course will be omitted November 11 because of Armistice Day.

DEATHS

DRAPER — CHARLES R. DRAPER, M.D., of 37 Forest Street, Medford, died October 25. He was in his seventy-sixth year.

A native of Vermont, he attended Medford High School and received his degree from the University of Vermont College of Medicine in 1888. After a postgraduate course at Columbia University College of Physicians and Surgeons, he practiced in St. Albans, Vermont, and later went to Medford.

Dr. Draper was a member of the Massachusetts Medical Society and the American Medical Association. Formerly a staff member of the Boston Dispensary he had been on the staff of the Lawrence Memorial Hospital in Medford since 1924, and had served as chairman of the medical board for the past six years. Dr. Draper was a former city physician of Medford and had also served for thirty years as physician for the Medford Home for the Aged.

His widow and a daughter survive him.

GLASS — JAMES GLASS, M.D., of 154 Union Avenue, Framingham, died July 4. He was in his sixty-eighth year.

Dr. Glass received his degree from the Harvard Medical School in 1904. He held memberships in the American Medical Association and the Massachusetts Medical Society. He served during the World War and was formerly on the staff of the Framingham Union Hospital.

HOBGOOD — Legan H. Hobgood, M.D., of New Bedford, died October 27. He was in his forty-fifth year.

A native of Tarboro, North Carolina, he graduated from Wake Forest College in Raleigh, North Carolina, and received his degree from the University of Maryland School of Medicine and College of Physicians and Surgeons in 1921. Dr. Hobgood served his internship in St. Luke's Hospital, New Bedford, and started practice there. After eight years he studied at the Brooklyn Eye and Ear Hospital and returned to his practice in New Bedford.

Dr. Hobgood was a member of the American Medical Association and the Massachusetts Medical Society.

MISCELLANY

CONNECTICUT NEWS

A Quack DeLuxe Convicted

"Doctor" Arthur Cere of Manchester, New Hampshire, was recently arrested in New Britain, Connecticut, and fined two hundred dollars for practicing medicine without a license. This quack was reported to be holding clinics at regular intervals in that city. His qualifications consisted entirely in his double and triple joints, together with the fact that he was the son of a seventh son and born under a veil, known in the old vernacular as a caul. He had supplemented his natural proficiency with some reading in books obtained through correspondence courses and was said to have taken a one-year course in a so-called "medical school" in Chicago, but he obtained most of his knowledge from a manual published by a nationally known patent medicine company. An investigation of his records revealed several hundred patients in Connecticut, Massachusetts, Maine and Vermont, and according to the diagnoses jotted beside the names his guesswork in medicine was limited largely to three diseases — those of the feet, high blood pressure and gastric ulcers, the last appearing to be the most popular.

Change in Test for Syphilis

On August 15 the Bureau of Laboratories of the State Department of Health substituted the Hinton test for the Kahn precipitation test in the routine examination of blood specimens for syphilis. This change was effected after a careful consideration of the results of the last two evaluation studies conducted by the United States Public Health Service with a large number of state health department laboratories participating. These studies furnished convincing evidence that a higher percentage of cases of syphilis are detected by the Hinton test than by any other test. It is of interest to note that Connecticut has finally fallen in line with Massachusetts in recognizing the value of the Hinton test.

Marriage License Law Specimens

During the first half of 1938, 7340 specimens of blood were examined by the Bureau of Laboratories of the State Department of Health for Marriage License Law purposes, in addition to a number of tests in other approved laboratories. Of these 106 were positive, 4 showed a questionable reaction, and the remainder indicated freedom from syphilis in the applicant.

Norwalk Adopts Full-Time Health Service

By vote of the city council in August the City of Norwalk placed the health officer on a full-time basis, effective September 1. Norwalk thus becomes the ninth city in Connecticut to have a full-time department of health. Because of his many years of successful experience, the mayor and council named Robert E. Perdue, M.D., as health officer for a term of four years. A public-health nurse and a clerk are to be added to the staff. The city is to be reimbursed for a portion of its new appropriation from United States Public Health Service funds.

Health Officer Appointments

W. Sherman Randall, M.D., has been appointed health officer of Shelton to complete the unexpired term left vacant by the death of J. Eugene Black, M.D.

Donald McCrann, M.D., has been appointed health officer of Bloomfield, replacing Maurice Thompson, M.D., who has moved to New York City to engage in full-time medical insurance work.

Archibald M. Gaulocher, M.D., has been appointed health officer of Kent, and Frank D. Ursone, M.D., of Norfolk, health officer of Colebrook.

Edward D. Brophy, M.D., has been appointed health officer of Norwich to complete the unexpired term of the late Harrison Gray, M.D.

Statistical Analysis of Cancer in Hartford

The Hartford Board of Health has published a statistical analysis of cancer from 1920-1936 in the form of a 60-page paper-covered booklet. This document is an interesting and valuable addition to the records of health work in the field of cancer. It furnishes many comparisons between statistics from New Haven and those from Hartford and furnishes data on the cancer mortality among the foreign born and native born and among the various age groups. During these seventeen years Hartford had 2905 deaths from malignant neoplastic disease among its residents.

Infant Mortality in Hartford

During the period 1931-1938 there has been a 60 per cent reduction in the infant mortality rate in Hartford. This is a greater reduction than occurred in the previous forty years. The death rate of infants from pneumonia has dropped more rapidly than the general pneumonia rate, and diarrhea has become an unimportant cause of infant mortality. The infant mortality rate for the first six months of 1938 was 30 deaths per 1000 live births.

Southington Opens New Hospital

On July 7 state and local officials, trustees of the Hospital Corporation of Southington and townspeople joined in dedicating the Bradley Memorial Hospital which will house the "Southington Plan" in caring for the public health needs of the community. The cost of the building, without furniture or hospital equipment, was about $100,000. It was built and equipped and will be maintained in part by a $200,000 fund provided in the will of Mrs. Julia Arnold Bradley who died in 1919.

Chief of Crippled Children's Division Resigns

Dr. Russell V. Fuldner, chief of the Division of Crippled Children in the State Department of Health, has resigned to accept a residency in orthopedic surgery at the

Massachusetts General Hospital. The division now conducts six clinics — at the Newington Home and in the general hospitals of Stamford, Danbury, Norwich, Willimantic and Derby.

Hospital Service Insurance in Connecticut

Hartford has just offered a plan of hospital service insurance to the public. This plan is in effect the same as the plan now in operation in New Haven, definitely excluding the services of any physician except interns and residents. This state now has group hospital insurance plans in operation in all the large cities of the western and southwestern sections. A committee of twenty-two has been appointed by Governor Cross to study the problem with a view to establishing a uniform plan throughout the State. As a corollary to this the Connecticut State Medical Society, through its Council, is investigating the practicability of sickness insurance whereby the patient is provided with funds with which to pay his physician.

Deaths

BELL — George N. Bell, M.D., aged sixty-eight, for forty-five years a surgeon in Hartford, died at his home in West Hartford on September 10. Dr. Bell had retired from active practice ten years ago because of ill-health. His death was said to have been caused by a cerebral hemorrhage. Prior to his retirement Dr. Bell had been a member of the surgical staff of the Hartford Hospital, becoming a consultant at the time of his retirement. A graduate of Yale University School of Medicine in 1892, he interned a year at the Hartford Hospital before engaging in active practice. He was born in Blandford, Massachusetts, son of the late Dr. N. S. Bell and Mary Hatch Bell. The family moved to Windsor shortly after his birth. Dr. Bell was a member of the New England Surgical Society and the American College of Surgeons. He is survived by a sister, Mrs. William Bailey, of California.

CROWLEY — William H. Crowley, M.D., aged seventy-one, for the past forty-nine years a practicing physician in Hartford, a director and staff member of St. Francis Hospital, died of heart disease on September 12. He was born in Hartford on April 6, 1867, son of the late Timothy and Mary Holmes Crowley. He graduated from the Buffalo University Medical School in 1890. Dr. Crowley then settled in Collinsville where he became active in town affairs, serving for a time as chairman of the Board of Education and as health officer in the town of Canton. He is survived by his widow, two children, a sister and three grandchildren.

FOX — Myron J. Fox, M.D., aged thirty-seven, of Waterbury, died at the Massachusetts General Hospital on June 28. He had been taken to Boston but a few days before his death. A native of Boston, he was graduated from Harvard, McGill and Johns Hopkins universities. He interned at the Waterbury Hospital, became house physician and then began practice in that city.

RICHMAN — Raynault D. Richman, M.D., aged fifty-four, surgical adviser to the Aetna Life Insurance Company for eighteen years, a retired lieutenant-colonel of the Connecticut National Guard, died of heart disease at his home in West Hartford on September 16. He retired from the Connecticut National Guard on February 4, 1935, because of an eye injury received two years previously in an automobile accident. His World War record includes service overseas; he enlisted as a first-lieutenant and was discharged at the end of the war as a captain. Dr. Richman was born in Morton, New York, son of Dr. Alonzo Richman and Sarah Dobson Richman. His grandfather was Dr. John Richman. He is survived by his widow, a son and a brother.

ROWELL — Edward E. Rowell, M.D., aged eighty-four, a practicing physician for almost sixty years, died at his home in Stamford on June 27 after an illness of a few weeks. He continued to practice up to his last illness. Dr. Rowell was born in West Concord, Vermont. He went to Stamford in 1873. He was a graduate of New York Homeopathic College and interned at Flower Hospital, New York City. Before going to Stamford he practiced for a short time at North Stratford, New Hampshire. He is survived by his widow and three children.

WAITE — Frank L. Waite, M.D., aged seventy-two, one of the leading ophthalmologists in Connecticut, died at his home in Hartford, September 1. His death followed a cerebral hemorrhage. Dr. Waite had relinquished active practice a few years ago, at the same time resigning as ophthalmologist at the Hartford Hospital to become a member of the consulting staff. He is survived by his widow.

WHITEHOUSE — Henry H. Whitehouse, M.D., aged seventy-four, of New York City, a native of Hartford and a prominent dermatologist and syphilologist, died at his summer home in Mystic, Connecticut, August 24. Dr. Whitehouse was a former president of the medical board of the New York Skin and Cancer Hospital. He had been instructor in several medical schools and had written numerous scientific articles. In his junior year at the Hartford Public High School he won the Connecticut scholarship on which he entered the Sheffield Scientific School of Yale University. He was graduated in 1885 with a degree of Bachelor of Philosophy and the next year engaged in research at the Yale physiological chemistry laboratory. He was graduated from the Columbia University College of Physicians and Surgeons in 1889. Early in his career Dr. Whitehouse became assistant in private practice to Dr. L. Duncan Bulkeley, an attending physician of the New York Skin and Cancer Hospital. He was instructor in dermatology at Cornell University Medical School from 1902 to 1907 and professor of dermatology and syphilology at Post Graduate Medical School and Hospital from 1914 to 1925. Dr. Whitehouse had been president of the New York Dermatological Society several times and was a diplomat of the American Board of Dermatology and Syphilology. Recently he was elected an honorary member of the American Dermatological and Syphilological Society.

CORRESPONDENCE

APPLICATIONS FOR FELLOWSHIP

To the Editor: I note in the October 20 issue of the *Journal* that thirty-five, or twenty-two per cent, of the applicants for fellowship in the Massachusetts Medical Society are graduates of diploma mills or inferior schools. What can we do to prevent the recognition of these inadequately trained men? Massachusetts still stands at the bottom of the list in the standards of medical practice. It is time that we act.

Earle M. Chapman, M.D.
270 Commonwealth Avenue,
Boston, Massachusetts.

NOTICES

BOSTON CITY HOSPITAL

The monthly clinicopathological conference will be held at the Boston City Hospital on Wednesday, November 9, at 12 o'clock noon in the Pathological Amphitheater.

JOSEPH E. HALLISEY, M.D., *Secretary*,
Medical Staff.

NEISSERIAN MEDICAL SOCIETY

The seventh annual meeting of the Neisserian Medical Society of Massachusetts will be held in the Hotel Kenmore, Boston, on November 16, at 8:15 p. m. The meeting will be preceded by a dinner at 6:30 p. m.

Dr. Adolph Jacoby, medical supervisor of the syphilis and gonorrhea clinics of the New York City Department of Health, will speak on "Sulfanilamide in the Treatment of Gonorrhea."

All who are interested are cordially invited to attend.

OSCAR F. COX, M.D., *Secretary*.

HARVARD MEDICAL SOCIETY

The next meeting of the Harvard Medical Society will be held on Tuesday, November 8, in the Peter Bent Brigham Hospital amphitheater (Shattuck Street entrance) at 8:15 p. m.

PROGRAM

Presentation of cases.

Recent Developments in the Treatment of Neurological Diseases: Epilepsy, hydrocephalus, athetosis and paralysis agitans. Dr. Tracy J. Putnam.

Medical students and physicians are cordially invited to attend.

ROBERT ZOLLINGER, M.D., *Secretary*.

TUMOR CLINIC, BOSTON DISPENSARY

Each Tuesday and Friday morning from ten to twelve-thirty there is a meeting of the Tumor Clinic of the Boston Dispensary, a unit of the New England Medical Center. All kinds of tumors are seen, discussed, and when indicated, treated with radium and high-voltage x-ray.

Physicians are welcome to visit this clinic and bring patients for diagnosis.

DEDICATION OF THE CHEEVER AMPHITHEATER

The new amphitheater in the Dowling Building at the Boston City Hospital will be dedicated to Dr. David Williams Cheever on Monday, November 7, at noon. Mr. Carl Dreyfus, Dr. Fred B. Lund and Dr. David Cheever will speak. Members of the profession are cordially invited to attend the exercises.

SOCIETY MEETINGS AND CONFERENCES

CALENDAR OF BOSTON DISTRICT FOR THE WEEK BEGINNING MONDAY, NOVEMBER 7

MONDAY, NOVEMBER 7

*12 m. Dedication of the Cheever Amphitheater, Dowling Building, Boston City Hospital.

*4 p. m. Physicians and medical students are cordially invited to attend a clinic presented by the medical, surgical and orthopedic services of the Infants' and Children's hospitals, in the amphitheater of the Children's Hospital.

TUESDAY, NOVEMBER 8

*9-10 a. m. Boston Dispensary. Progress in Diagnosis and Treatment of Malignant Diseases. Dr. W. M. Shedden.

*10 a. m. - 12:30 p. m. Tumor clinic. Boston Dispensary.

*8:15 p. m. Harvard Medical Society, Peter Bent Brigham Hospital amphitheater (Shattuck Street entrance).

WEDNESDAY, NOVEMBER 9

*9-10 a. m. Boston Dispensary. Hospital case presentation. Dr. S. J. Thannhauser.

*12 m. Clinicopathological conference. Children's Hospital amphitheater.

12 m. Boston City Hospital, clinicopathological conference. Pathological amphitheater.

*8:30 p. m. Boston Gastrointestinal Society, Beth Israel Hospital auditorium.

THURSDAY, NOVEMBER 10

8:30-9:30 a. m. Exchange visit, Surgical and Orthopedic Staffs of the Peter Bent Brigham and Children's hospitals, held this week at the Peter Bent Brigham Hospital.

*9-10 a. m. Boston Dispensary. Clinical Problems in Drug Addiction. Dr. Lowrey Davenport.

*10:30 a. m. Psychiatric Rounds. Boston City Hospital. Rounds will start at Room 916 on the ninth floor of the Medical Building.

SATURDAY, NOVEMBER 12

*9-10 a. m. Boston Dispensary. Hospital case presentation. Dr. S. J. Thannhauser.

*10 a. m. - 12 m. Staff rounds at the Peter Bent Brigham Hospital. Conducted by Dr. Henry A. Christian.

*Open to the medical profession.

NOVEMBER 4 — Joint meeting of the Boston Health League and the Boston Council of Social Agencies. Page 682, issue of October 27.

NOVEMBER 5 — American Board of Obstetrics and Gynecology. Page 145, issue of July 28.

NOVEMBER 5, 1938; FEBRUARY 4, MAY 15 and 16, 1939 — American Board of Obstetrics and Gynecology. Page 451, issue of September 22.

NOVEMBER 7 — Dedication of the Cheever Amphitheater. Notice above.

NOVEMBER 8 — Harvard Medical Society. Notice above.

NOVEMBER 9 — Boston City Hospital, clinicopathological conference. Notice above.

NOVEMBER 9 — Boston Gastrointestinal Society. Page 682, issue of October 27.

NOVEMBER 10 — Pentucket Association of Physicians, 8:30 p. m., Hotel Bartlett, 95 Main Street, Haverhill.

NOVEMBER 15-16 — New England Postgraduate Assembly. Page vii, advertising section, issue of September 29.

NOVEMBER 16 — Neisserian Medical Society of Massachusetts. Notice above.

JANUARY 12, 1939 — Peter Bent Brigham Hospital. Clinic conducted by Dr. Christian. Page 633, issue of October 20.

MARCH 27-31 — American College of Physicians. Page 36, issue of July 7.

MAY 7-15 — International Congress of Military Medicine and Pharmacy. Page 501, issue of September 29.

MAY 15-19 — American Medical Association, St. Louis, Missouri.

JUNE 6, 7, 8 — Massachusetts Medical Society. Worcester.

SEPTEMBER — Boston Psychoanalytic Institute. Page 450, issue of September 22.

DISTRICT MEDICAL SOCIETIES

ESSEX SOUTH

DECEMBER 7 — Salem Hospital. Clinic at 5 p. m. Dinner at 7 p. m. Speakers: Dr. Walter G. Phippen and Dr. Channing Frothingham. Subject: Economic Problems.

JANUARY 4 — Danvers State Hospital. Clinic at 5 p. m. Dinner at 7 p. m.

FEBRUARY 8 — Essex Sanatorium, Middleton. Clinic at 5 p. m. Dinner at 7 p. m. Speaker: Dr. Edward Churchill.

MARCH 1 — Lynn Hospital. Clinic at 5 p. m. Dinner at 7 p. m. Speaker: Dr. John Rock. Subject: Endocrinology.

APRIL 5 — Addison Gilbert Hospital, Gloucester. Clinic at 5 p. m. Dinner at 7 p. m. Speaker: Dr. Ethan Allan Brown. Subject: Allergy.

MAY 10 — Annual meeting. Salem Country Club, Peabody.

SUFFOLK

NOVEMBER 30 — Program of clinical and investigative activities. Staff of the Massachusetts General Hospital, Boston Medical Library, 8:15 p. m.

JANUARY 25, 1939 — Symposium on Diabetes. Dr. Elliott P. Joslin and associates, Boston Medical Library, 8:15 p. m.

MARCH 29 — Joint meeting with New England Pediatric Society, Boston Medical Library, 8:15 p. m. Program and speakers to be announced.

APRIL 26 — Annual meeting in conjunction with Boston Medical Library, at 8:15 p. m. Election of officers. Program and speakers to be announced.

BOOK REVIEWS

Pediatric Surgery. Edward C. Brenner. 843 pp. Philadelphia: Lea & Febiger, 1938. $10.00.

The author has been at great pains to present in condensed form a very wide field of surgery. The advances in surgery of infancy and childhood have been so great in the last decade that the reviewer is impressed with the great difficulty of producing in a single volume a work that can adequately cover this field. He agrees with the recent remark of a British reviewer who said that the surgeon of the present and future must turn more and more to monographs on special surgical subjects for his up-to-date information.

The chief criticism of this volume is that many of the author's advocated forms of treatment have already been superseded, as reference to the recent literature would show. The absence of any adequate bibliography, to which the interested surgeon might refer, further limits the value of the volume.

The Relationship Between Characteristics of Personality and Physique in Adolescents. P. S. de Q. Cabot. 120 pp. Provincetown: The Journal Press, 1938. $1.50.

For purposes of review this monograph may be divided into two parts. The first is a survey of the main investigations dealing with constitutional types. This part is excellent; it gives a brief but adequate summary of most of the important work done in this ever-new, ever-old, and ever-recurring subject. From the time of Hippocrates, physicians and others concerned with the bodily and mental health of man have endeavored to correlate physical type with the liability to disease and with temperament. In recent years this has become a problem of major importance, and certain distinguished names are associated with the studies. The list of writings as given by Cabot omits one important American investigator, William Washington Graves. It lays especial stress upon the work of Kretschmer whose famous pyknic, leptosome and athletic types were correlated by him with the liability to mental disease and especially to the manic-depressive or cyclothymic disorders and to schizophrenia, its antecedents and correlates, which lumped together may be called the schizothymic temperament.

European investigators have, on the whole, tended to confirm the work of Kretschmer, but as is pointed out by Cabot, good statistical and scientific methodology has been lacking. American investigators as a whole have not agreed with his findings in any absolute sense, although agreeing that the division of mankind into these physical types is of some help.

The original work, part two of the monograph, relates to Cabot's proposal to test the Kretschmerian hypothesis that with the pyknosomic build — the round, more or less fat, type of physique — there is associated a cyclothymic type of temperament and that with the leptosomic — asthenic — and athletosomic bodily build there is associated a schizothymic type of temperament. His investigation deals with the adolescent stage of development. The material consisted of 212 adolescent boys who had already been investigated in the Harvard Growth Study.

One can very readily and perhaps too easily criticize the methodology which consists, first, in a miscellaneous series of psychological investigations of the laboratory type concerning temperament, all of which have grave defects; second, a rating method by teachers concerning such matters as emotional sensitiveness to the environment, social introversion, extraversion, cyclothymic trends, schizothymic trends, competitiveness, talkativeness, intellectual leadership and social leadership. The subjects were likewise supposed to be able to rate themselves in respect to competitiveness, emotional sensitiveness to environment and social introversion and extraversion and to answer such questions as: "Are you ever worried by the thought that you are inferior to anybody?" "If a department store had promised to deliver a pair of football shoes in time for a football game but hadn't done so, would you be determined to tell the store clerk or anyone connected with the store how very dissatisfied you were with the service, or would you let it go?" To the reviewer it seems that the discrepancy between what a person says he will do in a questionnaire and what he will do when confronted with a real-life situation is so great as to make this method of almost no value in determining such matters as are involved in such a study.

However, Cabot says that on the whole there is little specific evidence to support Kretschmer in his contention that these various physical types are correlated with cyclothymic and schizothymic temperaments. He further states: "However, we have discovered a remarkable degree of association between the athletosomic bodily build and certain personality traits that, as a constellation, have high social value. The athletosomic physique is one that is most desirable from the viewpoint of social approval, physiological functioning, freedom of movement, resistance to stress and strain, aesthetics and general constitutional well-being. Thus, considered as having a biologically and socially 'good' physique, the athletosome, in relation to the average leptosome (asthenic) is more ascendant, extraverted, responsible, influential, and a better social leader. This is not what is expected in terms of Kretschmer's theory. Compared with the pyknosome, the athletosome is more ascendant, creative and imaginative, and less socially introverted."

This glorification of the athletosome probably has considerable validity in an American high school. An athletic body is an asset, but that all the virtues ascribed by Cabot to the athletosome inevitably appear in maturity seems very unlikely. There are probably great ranges of social activity in which the athletosome is not particularly represented. It is not likely that he is especially important in art or in the greatest intellectual work or that he is even in any degree the leader of nations. (Witness the present crop of dictators!) In a high school the athlete is the king and so he is in college. Later on, he tends to fade out from leadership. He is probably more apt to become a policeman than a professor, an aviator than an inventor.

The monograph is recommended highly to those who are interested in this very important subject. Fine scholarship is amply demonstrated, and a real attempt at a scientific approach to the problem has been made. The reviewer believes that the monograph really shows that as yet we have no yardsticks which adequately measure that confusing mosaic of qualities, the variable human being.

Handbook of Histological and Cytological Technique. R. R. Bensley. 167 pp. Chicago: The University of Chicago Press, 1938. $2.00.

One has hoped for some time that Professor Robert Bensley of the University of Chicago would make available his valuable knowledge of histological and cytological technics. This has now occurred in the appearance of this interestingly bound handbook. In the preface the author, in collaboration with Sylvia H. Bensley, states that "for the past twenty-five years the major attacks on problems of minute anatomy have been in regard to the cell

not so much as an integer of tissue structure and function but in the study of component and structural variation. This study of the integer of cells is revealed in the methods and techniques devised, developed, or adopted in this Department which we have collected in this volume." In view of the fact that the continued advance of knowledge is dependent on the development of ever-new technics, students and teachers will appreciate the significance of making available this material which Professor Bensley and his students have developed during the past twenty-five years. Beginners in this field will be especially delighted with this book because principles underlying the methods are consistently emphasized.

The text is divided into two parts, the first dealing with technics for the study of fresh tissues, and the second with those for the study of fixed tissues. Under the first, consideration is given to methods for studying living cells, surviving cells and dead cells and to the Altmann-Gersh freezing-dying method. The second part is subdivided under the subordinate headings of important general rules for laboratory technic, fixation, imbedding, staining, general tissue stains, blood stains, staining of cell constituents and cell inclusions, selective stains, neurohistological methods, visible microchemical reactions, the freezing microtome, decalcification, and miscellaneous technics.

Space does not permit going into the detailed consideration of any one of these parts or its subdivision, but it may be said that this handbook will undoubtedly find its place in every laboratory dealing with the various phases of biology.

The Infant: A handbook of modern treatment. Eric Pritchard. 744 pp. Baltimore: William Wood & Co., 1938. $6.00.

This volume, by one of the leading pediatricians in England, is based on a series of lectures which are given to postgraduate students at the Infants' Hospital in London.

It is primarily a handbook on "treatment" and written from the point of view of the general practitioner. It covers the whole range of the diseases of children under five years of age and expresses clearly the procedures used by the author, based on many years of practice. One is impressed with the reliance which the author places on drugs in influencing pathologic processes. Many of the procedures advocated have been abandoned in this country.

The book provides an interesting review of a wide range of therapeutic procedures, but the reviewer questions the value of many of these. There is no discussion of diagnosis or of pathologic anatomy.

Clinics on Secondary Gastro-Intestinal Disorders; Reciprocal Relationships. Julius Friedenwald, Theodore H. Morrison and Samuel Morrison. 251 pp. Baltimore: William Wood & Co., 1938. $3.00.

This work by a group of gastroenterologists headed by Julius Friedenwald is novel in that it is devoted to the study of digestive symptoms due not to lesions in the gastrointestinal tract but to abnormalities in other organs. It is a truism that disturbances of the gastrointestinal tract may frequently have ill effects on the body as a whole. What the authors stress is that disease processes elsewhere in the body may produce such marked secondary effects on the gastrointestinal tract that the digestive symptoms overshadow those produced directly by the primary affection. Thus a patient suffering from angina pectoris may suffer from distention, flatulence and epigastric constriction and be treated for indigestion, with the true condition overlooked.

The authors teach a wholesome outlook in medicine when they stress that there are few diseases which are not accompanied by gastrointestinal manifestations of one sort or another and that on this account it is extremely important, whenever gastrointestinal symptoms arise, to institute a complete general survey of the entire organism to discover every possible etiologic factor. This is applicable to the entire field of clinical medicine.

The book is arranged in the form of clinics, each taking up the affections of an organ or system in which gastrointestinal symptoms are commonly prominent. The list is as follows: (1) cardiac diseases, (2) pulmonary tuberculosis, (3) liver and gall-bladder diseases, (4) intestinal diseases, (5) focal infections, (6) genitourinary diseases, (7) diseases of the female pelvic organs, (8) menopause, (9) endocrine dysfunctions, (10) syphilis, (11) hematopoietic and deficiency disturbances, (12) nervous affections, (13) skin diseases, (14) eye diseases and (15) food allergy.

This work presents an important point of view and will prove valuable alike to students, general practitioners and specialists.

The Principles and Practice of Medicine: Designed for the use of practitioners and students of medicine. Originally written by Sir William Osler and revised by Henry A. Christian. Thirteenth edition. 1424 pp. New York and London: D. Appleton-Century Co., 1938. $9.00.

Eight years have passed since the last edition of this famous textbook was published. The revision of four previous editions had been carried on by Thomas McCrae, who has since died. Now this important labor has been taken over by Henry A. Christian, the Hersey Professor of the Theory and Practice of Physic, Harvard University. He has assumed responsibility for all changes and additions not credited by name to some other individual. He has seen fit, fortunately, to keep the standard form set by Osler, so that the revision is comparatively unobtrusive and one feels that the old book, so well known to more than a generation of students, is still the most beloved as well as the best single-volume textbook of medicine ever written in English. The additions have been carefully done, and the reviewer finds nothing to criticize in the way that the Osler tradition has been carried on by the present editor. The new material is timely, and the book can be highly recommended.

Subacute and Chronic Pericardial and Myocardial Lesions Due to Non-Penetrating Traumatic Injuries. Erik Warburg. 147 pp. New York and London: Oxford University Press, 1938. $6.00.

This is a collection of all the case reports in the world literature which the author considered fitted the picture of traumatic blunt injury to the heart. A total of 203 cases is included. The critical reviewer would, however, exclude the majority of the cases as being either doubtful or due to some unrelated condition such as tuberculous pericarditis, coronary disease or cardiac neurosis. A biographical sketch of Orluff Borch, who first described this condition in 1676, is appended.